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NOTICE.

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The Council think it proper to state that the authors of the several communications are alone responsible for the statements, reasonings, and opinions contained in their respective papers.

20, Hanover Square, W.;
October 9th, 1903.

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1889 Bisshopp, Francis Robert Bryant, M.A., M.B., B.S., Belle Vue, Mount Pleasant, Tunbridge Wells.

1893 Bond, James William, M.D., 26, Harley Street, W. *C.C.* 2.

1898 Bosanquet, William Cecil, M.D., 117a, Harley Street, Cavendish Square, W.

1888 Bostock, Robert Ashton, Cefn Mor, Penmaen, Glamorganshire.

1900 Bousfield, Arthur, M.B., The Cheal, Hornsey Lane, N.

1899 Bowlan, Marcus Marwood, M.B., B.S., The Infirmary, Raine Street, St. George-in-the-East, E.

1883 Bowlby, Anthony A., C.M.G. (*Hon. Secretary*), 24, Manchester Square, W. (C. 1891–3, S. 1901– .) *Trans.* 5, *C.C.* 4.

1883 Bowles, Robert Leamon, M.D., 16, Upper Brook Street, W. (C. 1890–1.)

1896 Box, Charles R., M.D., 2, Devonshire Place, Portland Place, W. *Trans.* 1.

1889 Boyd, Stanley, B.S., 134, Harley Street, W. (C. 1899–1902.) *Trans.* 1, *C.C.* 7.

1890 Bradford, John Rose, M.D., F.R.S., 8, Manchester Square, W. (C. 1900–1902.) *Trans.* 2.

1878 Bridges, Robert, M.B., M.A., The Manor House, Yattendon, Berkshire. *Trans.* 1.

1868 Bright, George Charles, M.D., Cannes, Alpes Maritimes, France.

O.M. Broadbent, Sir William Henry, Bart., K.C.V.O., M.D., F.R.S., 84, Brook Street, W. (C. 1871–3, V.P. 1881–3, P. 1887–8.) *Trans.* 17.

1887 Brock, J. H. E., M.D., B.S., 77, Fellows Road, South Hampstead, N.W.

1894 **Brodie**, C. Gordon, Fernhill, Wootton Bridge, Isle of Wight. *C.C.* 1.

1898 **Brook**, William Henry Breffit, M.D., B.S., 33, Silver Street, Lincoln. *Trans.* 3.

1890 Brown, Walter Henry, 19, Queen Street, Leeds. *Trans.* 2.

ELECTED

1876 BROWNE, GEORGE BUCKSTON, 80, Wimpole Street, W. (C. 1891–3.) *Trans.* 3.

1893 BROWNE, JAMES WILLIAM, M.B., 37, Holland Park Avenue, W. *Trans.* 1.

1887 BROWNE, OSWALD AUCHINLECK, M.A., M.D., 7, Upper Wimpole Street, W.

1883 BRUCE, JOHN MITCHELL, M.D., 23, Harley Street, W. (C. 1901– .)

1893 BRYANT, JOHN HENRY, M.D., 4, St. Thomas's Street, S.E., and 8, Mansfield Street, Portland Place, W. *Trans.* 2.

O.M. BRYANT, THOMAS, M.Ch., 27, Grosvenor Street, W. (C. 1872, V.P. 1876–7, P. 1885–6.) *Trans.* 9.

1890 BUCKLAND, FRANCIS O., B.A., M.B., C.M., Brockenhurst, Hants. *Trans.* 1.

1901 BUCKNALL, THOMAS RUPERT HAMPDEN, M.S., 35, Harley Street, W.

1891 BURGHARD, FREDERIC FRANÇOIS, M.D., M.S., 86, Harley Street, W.

1881 BURNET, ROBERT WILLIAM, M.D., 36, Grosvenor Street, W. *Trans.* 1.

1879 BURTON, WILLIAM EDWARD, 24, Wimpole Street, W.

1900 BUSCH, JOSEF PAUL ZUM, M.D., Chiswell House, 133, Finsbury Pavement, E.C. *Trans.* 1.

1881 BUTLIN, HENRY TRENTHAM, 82, Harley Street, W. (C. 1887–9.) *Trans.* 7, *C.C.* 1.

1884 BUXTON, DUDLEY WILMOT, M.D., B.S., 82, Mortimer Street, W.

1902 BUZZARD, EDWARD FARQUHAR, M.B., 33, Harley Street, W.

O.M. BUZZARD, THOMAS, M.D., 74, Grosvenor Street, W. (S. 1870–2, C. 1873–6, V.P. 1880–1, P. 1895–7.) *Trans.* 15, *C.C.* 3.

1901 CADDY, ADRIAN, M.B., St. George's Hospital, Hyde Park Corner, S.W.

1891 CADDY, ARNOLD, 2/2, Harington Street, Calcutta.

1893 CALEY, HENRY ALBERT, M.D., 24, Upper Berkeley Street, W. *C.C.* 1.

1890 CALVERT, JAMES, M.D., 113, Harley Street, Cavendish Square, W. *Trans.* 2, *C.C.* 2.

1893 CAMPBELL, HARRY, M.D., 23, Wimpole Street, W., and Couiston Cottage, Fox Lane, Upper Norwood, S.E. *C.C.* 4.

1891 CARLESS, ALBERT, M.S., M.B., 10, Welbeck Street, W.

1890 CARR, JOHN WALTER, M.D., 19, Cavendish Place, W. (C. 1903– .) *Trans.* 2, *C.C.* 6.

1901 CARSON, HERBERT WILLIAM, 26, Welbeck Street, Cavendish Square, W.

1901 Carwardine, THOMAS, M.S., 16, Victoria Square, Clifton, Bristol. *Trans.* 1.

1885 CATON, RICHARD, M.D., 86, Rodney Street, Liverpool. *Trans.* 3.

O.M. CAYLEY, WILLIAM, M.D., 27, Wimpole Street, W. (C. 1874–5, S. 1876–8, C. 1879–80, V.P. 1885–6.) *Trans.* 8, *C.C.* 3.

1890 CHAPMAN, CHARLES WILLIAM, M.D., 21, Weymouth Street, W. (C. 1900–3.) *Trans.* 2, *C.C.* 2.

1884 CHAPMAN, PAUL M., M.D., 1, St. John's Street, Hereford. *C.C.* 1.

1893 CHARSLEY, ROBERT STEPHEN, "The Old Barn," Slough.

ELECTED

1885 CHEYNE, W. WATSON, C.B., M.B., C.M., F.R.S., 75, Harley Street, W. (C. 1894–6.) *Trans.* 2, *C.C.* 8.

1873 CHISHOLM, EDWIN, M.D., 44, Roslyn Gardens, Darlinghurst, Sydney, New South Wales.

O.M. **CHURCH**, Sir WILLIAM SELBY, Bart., K.C.B., M.D., 130, Harley Street, W. (C. 1874–6.)

1873 CHURTON, THOMAS, M.D., 35, Park Square, Leeds. (C. 1889–91.) *Trans.* 6.

O.M. CLAPTON, EDWARD, M.D., Towercroft, 41, Eltham Road, Lee, S.E. (C. 1872–4.) *Trans.* 1.

1874 CLARK, ANDREW, 71, Harley Street, W. *Trans.* 1.

1887 CLARK, FRANCIS WILLIAM, Hong-Kong.

1898 CLARKE, JAMES JACKSON, M.B., 18, Portland Place, W. *Trans.* 1, *C.C.* 8.

1885 Clarke, J. MICHELL, M.D., 28, Pembroke Road, Clifton, Bristol.

1885 CLARKE, WILLIAM BRUCE, M.B., 51, Harley Street, W. (C. 1894–7.) *Trans.* 2, *C.C.* 3.

1877 Clay, ROBERT HOGARTH, M.D., 4, Windsor Villas, Plymouth.

1887 CLEMOW, ARTHUR HENRY WEISS, M.D., C.M., 101, Earl's Court Road, W. *Trans.* 1, *C.C.* 1.

1877 **CLUTTON**, HENRY HUGH, M.A., 2, Portland Place, W. (C. 1885–7, V.-P. 1902– .) *Trans.* 12, *C.C.* 3.

1898 COBB, ERNEST HERBERT, Belmont, Stevenage, Herts.

1893 COLE, ROBERT HENRY, M.D., Moorcroft, Hillingdon, Middlesex.

1882 COLLIER, HERBERT, M.D., The Grange, Gorleston, Great Yarmouth, Norfolk.

1902 COLLINS, VICTOR EVELYN, M.B., Graaff Reinet, Cape Colony.

1896 COLMAN, WALTER STACY, M.D., 9, Wimpole Street, W. (C. 1903– .) *Trans.* 2, *C.C.* 3.

1882 COLQUHOUN, DANIEL, M.D., Dunedin, New Zealand.

1868 COOPER, FRANK W. (address uncommunicated).

1899 COOPER, HARRY, M.D., Fownhope, Surbiton Hill, Surrey.

1903 COOPER, PERCY ROBERT, M.D., "Glenthorn," The Downs, Bowdon, Cheshire.

1902 CORNER, EDRED MOSS, B.Sc., M.B., 28, Harley House, Regent's Park, W. *Trans.* 1.

1880 COTTLE, WYNDHAM, M.D., 39, Hertford Street, W.

O.M. COUPER, JOHN, 80, Grosvenor Street, W. (C. 1874.)

1875 COUPLAND, SIDNEY, M.D., 16, Queen Anne Street, W. (S. 1882–4, C. 1885–7, V.-P. 1898–1901.) *Trans.* 5, *C.C.* 3.

1886 COUSINS, JOHN WARD, M.D., Riversdale, Kent Road, Southsea. *Trans.* 1.

1882 COXWELL, C. F., M.B., Brighton, Melbourne, Victoria. *Trans.* 2.

1897 CRAWFURD, RAYMOND H. P., M.D., 71, Harley Street, Cavendish Square, W. *C.C.* 3.

ELECTED

1879 CRIPPS, WILLIAM HARRISON, 2, Stratford Place, W. (C. 1886–8.) *Trans.* 3.

1872 CRITCHETT, Sir G. ANDERSON, 21, Harley Street, W.

1877 CROCKER, HENRY RADCLIFFE, M.D., 121, Harley Street, W. (C. 1884–5.) *Trans.* 16.

1896 CROOKE-LAWLESS, W. R., Surg.-Maj. A.M.S., M.D., Guards' Club, S.W.

1898 CROUCH, HERBERT CHALLICE, 31, Welbeck Street, Cavendish Square, W.

1893 CURTIS, HENRY JONES, M.D., B.S., 152, Harley Street, W. *C.C.* 1.

1902 CUTHBERT, CHARLES FIRMIN, 2, Barton Street, Gloucester.

1872 DALBY, Sir WILLIAM BARTLETT, M.B., 18, Savile Row, W. (C. 1879–81.) *Trans.* 4.

1896 DALTON, NORMAN, M.D., 4, Mansfield Street, Portland Place, W. *Trans.* 2, *C.C.* 7.

1898 DARLEY-HARTLEY, WILLIAM, M.D., Seapoints, Cape Town, South Africa.

1893 DAUBER, JOHN HENRY, M.B., B.Ch., 29, Charles Street, Berkeley Square, W.

1886 DAVIES, ARTHUR T., M.D., 23, Finsbury Square, E.C. (C. 1897–1900.) *C.C.* 13.

1879 Davy, HENRY, M.D., 29, Southernhay, Exeter.

1889 DEAN, HENRY PERCY, M.B., 69, Harley Street, W.

1897 DEANE, JOHN HENRY, 50, Wallwood Road, Leytonstone, Essex.

1899 DE CHAZAL, EDMOND LUCIEN, M.D., Port Louis, Mauritius.

1903 DELBRÜCK, RAOUL ERNEST, M.B., 13, Buckingham Gate, S.W.

1879 Dennis, FREDERIC S., M.D., 542, Madison Avenue, New York, U.S.A.

1875 DENT, CLINTON T., 61, Brook Street, W. (C. 1884–6.) *Trans.* 5. *C.C.* 1.

O.M. DICKINSON, WILLIAM HOWSHIP, M.D., 9, Chesterfield Street, W (C. 1874–5, V.P. 1888–9.) *Trans.* 2.

1891 DICKINSON, WILLIAM LEE, M.D., 9, Chesterfield Street, W. (C. 1902– .) *Trans.* 8, *C.C.* 1.

1894 Dickson, THOMAS HUGH, M.B., B.C., H.M. Customs, Lower Thames Street, E.C.

1871 DIVER, EBENEZER, M.D., 7, Pittville Terrace, Stamshaw Road, Portsmouth. (C. 1890–2.) *Trans.* 1.

1901 DOUGLAS, JOHN J., M.D., 42, Central Hill, Norwood, S.E.

1868 DRAGE, CHARLES, M.D., Hatfield, Herts.

1896 DREW, DOUGLAS, M.D., B.S., 49, Devonshire Street, Portland Place, W.

O.M. DUCKWORTH, Sir DYCE, M.D., LL.D. 11, Grafton Street, Bond Street, W. (C. 1875–7, V.P. 1887–8, P. 1891–2.) *Trans.* 20, *C.C.* 7.

1902 DUDGEON, LEONARD S., 6, Powis Gardens, Bayswater, W. *C.C.* 2.

1884 DUKE, EDGAR, 11, Wilbury Road, Hove.

1869 DUKE, OLLIVER THOMAS, M.B., Surgeon, Bengal Army, India.

1899 DUNCAN, ANDREW, M.D., 24, Chester Street, Grosvenor Place, S.W. *Trans.* 1.

Elected

1889 Duncan, John, M.D., St. Petersburg.
1889 Dunn, Louis Albert, M.S., 51, Devonshire Street, Portland Place, W. (C. 1899–1902.) *Trans.* 2, *C.C.* 2.
1884 Edmunds, Walter, M. C., 2, Devonshire Place, Portland Place, W. (C. 1902– .) *Trans.* 2.
1899 Eïd, Alfred, M.D., Cairo, Egypt.
1902 Elliot, Andrew, M.D., 11, Upper Berkeley Street, Portman Square, W.
1882 Emond, Emile, M.D., 113, Boulevard Beaumarchais, Paris.
1902 English, Thomas Crisp, 1, Park Crescent, Portland Place, W. *C.C.* 1.
1868 Evans, Julian, M.B., 123, Finborough Road, Redcliffe Square, S.W.
1893 Evans, Willmott H., M.D., B.S., 2, Upper Wimpole Street, Cavendish Square, W. *C.C.* 1.
1888 **EVE**, Frederic S., 125, Harley Street, Cavendish Square, W. (C. 1899–1902.) *Trans.* 5, *C.C.* 10.
1877 Ewart, William, M.D., 33, Curzon Street, W. (C. 1884–6.) *Trans.* 4, *C.C.* 4.
1899 Fagge, Charles Herbert, M.S., 22, St. Thomas's Street, London Bridge, S.E.
1868 Fairbank, Frederick Royston, M.D., Westcott, Dorking, Surrey. *Trans.* 2.
1889 Fardon, Edward Ashby, Middlesex Hospital, W.
1900 Fawcett, John, M.D., 26, St. Thomas's Street, London Bridge, S.E. *Trans.* 1.
1902 Fedden, Walter Fedde, M.B., Darenth Asylum, Dartford.
1885 Fenn, Edward Liveing, M.D., Nayland, Colchester.
1902 Fennell, Charles Henry, M.D., Darenth Asylum, Dartford. *C.C.* 1.
1887 Fenwick, E. Hurry, 14, Savile Row, W. *Trans.* 2, *C.C.* 2.
1872 Fenwick, J. C. J., M.D., Long Framlington, Morpeth.
1893 Fenwick, William Soltau, M.D., 29, Harley Street, W. *Trans.* 2.
1878 Field, George P., 34, Wimpole Street, W.
1876 Finlay, David White, M.D., 2, Queen's Terrace, Aberdeen. (C. 1885–7, S. 1891.) *Trans.* 4.
1894 Fletcher, Herbert Morley, M.D., 98, Harley Street, W. *Trans.* 1.
1878 **FONMARTIN**, Henry de, M.D. (Travelling.)
1889 Forbes, Daniel Mackay, 5, Bedford Place, West Croydon.
1890 Foster, Michael G., M.D., M.A., Villa Camilla, San Remo, Italy.
1898 Fowler, Charles Owen, M.D., Cotford House, Thornton Heath, Surrey.
1881 Fowler, James Kingston, M.D., 35, Clarges Street, W. (C. 1887–9, 1899–1902, S. 1896–9.) *Trans.* 4, *C.C.* 6.
1886 Fox, R. Hingston, M.D., 23, Finsbury Square, E.C. *C.C.* 1.
1878 Fox, Thomas Colcott, M.B., 14, Harley Street, W. (C. 1888–90.) *Trans.* 7, *C.C.* 2.
1893 Foxwell, Arthur, M.D., 22, Newhall Street, Birmingham.
1902 French, Herbert, M.B., 26, St. Thomas's Street, London Bridge, S.E.

ELECTED

1897 FRIPP, Sir ALFRED DOWNING, C.B., M.V.O., M.S., 19, Portland Place, W. *Trans.* 3.

1901 FRITH, WALTER S., M.B., The Limes, Rickmansworth, Herts.

1899 FÜRTH, KARL, M.D., 39, Harley Street, W.

1890 FULLER, HENRY ROXBURGH, M.D., 45, Curzon Street, W.

1897 FURNIVALL, PERCY, 28, Weymouth Street, Portland Place, W.

1891 FYFFE, W. KINGTON, M.B., B.C., 19, Duke Street, Manchester Square, W. (Travelling, New Zealand.) *Trans.* 1.

1888 GAGE-BROWN, CHARLES HERBERT, M.D., 85, Cadogan Place, S.W.

1895 GALLOWAY, JAMES, M.D., 54, Harley Street, W.

1887 GARROD, ARCHIBALD EDWARD, M.A., M.D., 9, Chandos Street, Cavendish Square, W. (C. 1898–1901.) *Trans.* 4, *C.C.* 3.

1879 GARSTANG, THOMAS WALTER HARROPP, Englefield, Delamer Road, Bowdon, Cheshire.

1903 GATES, EDWARD A., 11, Collingham Road, South Kensington, S.W.

1885 GIBBONS, ROBERT ALEXANDER, M.D., 29, Cadogan Place, S.W. *Trans.* 1.

1893 GIBBS, CHARLES, 115, Harley Street, W.

1875 GILBART-SMITH, T., M.A., M.D., 68, Harley Street, W. (C. 1883–5.)

1868 GLOVER, JAMES GREY, M.D., 25, Highbury Place, N. (C. 1878–80, V.P. 1892–4.) *Trans.* 2.

1893 GLOVER, LEWIS G., M.B., B.C., 17, Belsize Park, Hampstead, N.W.

1875 GODLEE, RICKMAN JOHN, M.S., 19, Wimpole Street, W. (C. 1882–3, 1887–9, S. 1884–6, V.P. 1895–8.) *Trans.* 14, *C.C.* 10.

1901 GOFFE, ERNEST GEORGE LEOPOLD, Western Hospital, Seagrave Road, Fulham, S.W.

1882 GOLDIE, ROBERT WILLIAM, Hirst House, Hirst, Morpeth, Northumberland.

1878 GOLDING-BIRD, C. H., M.B., 12, Queen Anne Street, W. (C. 1887–9, V.-P. 1903– .) *Trans.* 18, *C.C.* 1.

1894 GOODALL, EDWARD WILBERFORCE, M.D., Eastern Hospital, Homerton, N.E. *Trans.* 6.

1875 GOODHART, JAMES FREDERIC, M.D., 25, Portland Place, W. (C. 1880–2, V.P. 1897–1900.) *Trans.* 14, *C.C.* 2.

1891 GOODMAN, ROGER NEVILLE, M.B., Elmside, Kingston-on-Thames.

1869 GOODRIDGE, HENRY FREDERICK AUGUSTUS, M.D., 10, Brock Street, Bath.

1882 GOODSALL, D. H., 17, Devonshire Place, W.

1877 GOULD, A. PEARCE, M.S., 10, Queen Anne Street, W. (C. 1885–7, 1895–8, S. 1892–5, V.-P. 1898–1901.) *Trans.* 14, *C.C.* 9.

1901 GOWAN, BOWIE CAMPBELL, Raven Dene, Great Stanmore, Middlesex.

1875 GOWERS, Sir WILLIAM RICHARD, M.D., F.R.S., 50, Queen Anne Street W. (C. 1881–2, V.-P. 1903– .) *Trans.* 5, *C.C.* 1.

1891 GRANT, J. DUNDAS, M.D., 18, Cavendish Square, W.

1868 GREEN, T. HENRY, M.D., 74, Wimpole Street, W. (C. 1877–9.) *Trans.* 2, *C.C.* 1.

ELECTED

1875 Greenfield, WILLIAM SMITH, M.D., 7, Heriot Row, Edinburgh. (C. 1881.) *Trans.* 3.

1893 **GRIFFITH**, WALTER SPENCER ANDERSON, M.D., 96, Harley Street, W.

1895 Grube, KARL, M.D., Neuenahr, Germany. *Trans.* 1.

1895 GUTHRIE, LEONARD GEORGE, M.D., 15, Upper Berkeley Street, W. *Trans.* 1, *C.C.* 7.

1887 HABERSHON, SAMUEL HERBERT, M.D., 88, Harley Street, W. (C. 1901– .) *C.C.* 1.

1900 HADLEY, WILFRED J., M.D., 58, Harley Street, Cavendish Square, W.

1875 HALE, C. D. B., 3, Sussex Place, W. *Trans.* 1.

1903 HALL, JOHN BASIL, M.C., 31, Manningham Lane, Bradford.

1878 HALL, F. DE HAVILLAND, M.D., 47, Wimpole Street, W. (C. 1885–7.) *Trans.* 8, *C.C.* 5.

1889 HALSTEAD, GEORGE EZRA, M.D., B.S., Albion Hill House, Ramsgate.

1888 HANDFIELD-JONES, MONTAGU, M.D., 35, Cavendish Square, W.

1886 Handford, HENRY, M.D., 6, Regent Street, Nottingham. (C. 1893–4.) *Trans.* 8, *C.C.* 1.

1886 HARDIE, JAMES, M.D., 15, St. John Street, Manchester.

1890 HARPER, JAMES, M.D., 25, Rosary Gardens, South Kensington, S.W.

1872 HARRIS, HENRY, M.D., Trengweath, Redruth, Cornwall.

1889 HARRIS, HERBERT ELWIN, M.B., 13, Lansdown Place, Victoria Square, Clifton, Bristol. *Trans.* 1.

1881 HARRISON, CHARLES EDWARD, M.B., Grenadier Guards Hospital, Rochester Row, S.W.

1892 HARRISON, DAMER, 53, Rodney Street, Liverpool. *Trans.* 1.

1869 **HAWARD**, J. WARRINGTON, 57, Green Street, Grosvenor Square, W. (C. 1876–8, 1884–6, S. 1881–3, V.P. 1888.) *Trans.* 15.

1899 Hawkes, CLAUDE SOMERVILLE, Glencairn, Wickham Terrace, Brisbane, Queensland, Australia.

1886 HAWKINS, FRANCIS HENRY, M.D., 73, London Street, Reading. (C. 1902– .) *Trans.* 4, *C.C.* 3.

1889 HAWKINS, HERBERT PENNELL, M.D., B.Ch., 56, Portland Place, W. (C. 1900–3.) *Trans.* 1.

1890 HAWKINS-AMBLER, GEORGE ARTHUR, 30, Rodney Street, Liverpool.

1900 HAYWARD, JOHN ARTHUR, M.D., 31, Lingfield Road, Wimbledon, S.W.

1879 HENDERSON, GEORGE COURTENAY, M.D., Kingston, Jamaica, West Indies.

1884 HERRINGHAM, WILMOT PARKER, M.D., 40, Wimpole Street, W. (C. 1894–7.) *Trans.* 6, *C.C.* 3.

1897 HILLIER, ALFRED P., M.D., C.M., 30, Wimpole Street, W.

1874 HOLDERNESS, WILLIAM BROWN, 15, Park Street, Windsor.

1868 Holman, CONSTANTINE, M.D., 26, Gloucester Place, Portman Square, W. (C. 1894–7.)

ELECTED

1878 HOOD, DONALD WILLIAM CHARLES, M.D., 43, Green Street, Park Lane, W. (C. 1891–3.) *Trans.* 1.

1884 HORSLEY, Sir VICTOR, M.B., F.R.S., 25, Cavendish Square, W. (C. 1894.) *Trans.* 2, *C.C.* 1.

1901 HORTON-SMITH, PERCIVAL, M.D., 19, Devonshire Street, Portland Place, W.

1895 HOUGH, CHARLES HENRY, Full Street, Derby.

1880 **HOVELL**, T. MARK, 105, Harley Street, W. *Trans.* 1.

1893 HOWARD, R. J. BLISS, M.D., 31, Queen Anne Street, W.

1876 HOWSE, Sir HENRY GREENWAY, M.S., 59, Brook Street, W. (C. 1881–3, V.P. 1890–2.) *Trans.* 3.

1902 HULBERT, ERNEST BEDDOE, M.D., 77, Welbeck Street, Cavendish Square, W.

1898 HULKE, SYDNEY BACKHOUSE, Ivy House, Walmer, Kent.

1897 HUMPHRYS, CHARLES B., Eagle House, Blandford, Dorset.

1897 HUNT, GEORGE BERTRAM, M.B., 47, Albemarle Crescent, Scarborough. *Trans.* 1, *C.C.* 1.

1892 HUNTER, WILLIAM, M.D., 103, Harley Street, W.

O.M. HUTCHINSON, JONATHAN, F.R.S., 15, Cavendish Square, W. (C. 1867–8, V.P. 1875–6.) *Trans.* 14, *C.C.* 1.

1896 HUTCHINSON, JONATHAN, jun., 15, Cavendish Square, W. (C. 1902– .) *Trans.* 4, *C.C.* 5.

1893 IONIDES, THEODORE HENRY, M.B., B.S., 25, First Avenue, Brighton.

1883 JACKSON, GEORGE HENRY, Ashburton, Carew Road, Eastbourne.

O.M. JACKSON, J. HUGHLINGS, M.D., F.R.S., 3, Manchester Square, W. (C. 1872–3, V.P. 1887.) *Trans.* 2, *C.C.* 1.

1877 JACOBSON, WALTER HAMILTON ACLAND, M.B., M.Ch., 66, Great Cumberland Place, W. (C. 1890–2.) *Trans.* 2, *C.C.* 1.

1888 JAMES, JAMES THOMAS, M.D., 30, Harley Street, W.

1901 JOHNSON, EDWARD ANGAS, M.B., B.S., St. Catherine's, Prospect, South Australia.

1889 JOHNSON, RAYMOND, M.B., B.S., 11, Wimpole Street, W. (C. 1900–3.) *Trans.* 5, *C.C.* 4.

1893 JOHNSTON, G. F., M.D., 3, Montagu Place, Montagu Square, W.

1903 JONES, ERNEST LLOYD, 59, Trumpington Street, Cambridge.

1902 JONES, LAWRENCE, 63, Addison Road, W.

1898 Jones, ROBERT, 11, Nelson Street, Liverpool. *Trans.* 2.

1872 JONES, THOMAS RIDGE, M.D., 4, Chesham Place, S.W. (C. 1892–3.)

1876 JORDAN, FURNEAUX, Harborne, Birmingham. *Trans.* 1.

1902 JOWERS, REGINALD F., 55, Brunswick Square, Brighton.

Elected

1878 Keetley, Charles Robert Bell, 56, Grosvenor Street, W. (C. 1895–8.) *Trans.* 2.

1898 Kellock, Thomas Herbert, 8, Queen Anne Street, Cavendish Square, W. (C. 1903– .) *Trans.* 2, *C.C.* 4.

O.M. Kelly, Charles, M.D., Ellesmere, Gratwicke Road, Worthing, Sussex.

1901 Kelynack, Theo. N., M.D., 53, Harley Street, W.

1901 Keogh, Lieut.-Col. Alfred, C.B., M.D., 18, Victoria Street, S.W.

1897 Keyser, Charles Ralph, 29, Hamilton Terrace, N.W.

1883 Kidd, Percy, M.D., 60, Brook Street, W. (C. 1891–2, 1902– , S. 1899–1902.) *Trans.* 5, *C.C.* 10.

1887 Knaggs, R. Lawford, B.C., 27, Park Square, Leeds. *Trans.* 2.

1878 Lacey, Thomas Warner, 196, Burrage Road, Plumstead.

1897 Lamplough, Charles, Chatteris, Cambridgeshire.

1890 Lancaster, Ernest Le Cronier, M.B., B.Ch., Winchester House, Swansea, S. Wales. *Trans.* 2.

1895 Lane, James Ernest, 46, Queen Anne Street, W. *Trans.* 1.

1883 Lane, William Arbuthnot, M.B., M.S., 21, Cavendish Square, W. (C. 1893–6.) *Trans.* 29, *C.C.* 10.

O.M. Langton, John, 62, Harley Street, W. (C. 1878–80, V.P. 1892–4, P. 1897–9.) *Trans.* 5.

1886 Lankester, Herbert, M.D., Ingleborough, Woking, Surrey.

1885 Larder, Herbert, Whitechapel Infirmary, Vallance Road, E. *C.C.* 1.

1893 Lawson, Arnold, M.D., 12, Harley Street, W.

O.M. Lawson, George, 12, Harley Street, W. (S. 1871–3, C. 1874–6, V.P. 1881–3.) *Trans.* 16.

1900 Leaf, Cecil H., M.B., 75, Wimpole Street, Cavendish Square, W.

1877 Lediard, Henry Ambrose, M.D., 35, Lowther Street, Carlisle. (C. 1889, V.-P. 1902– .) *Trans.* 5, *C.C.* 1.

1896 Leech, Priestley, M.D., King Cross, Halifax.

1877 Lees, David B., M.D., 22, Weymouth Street, W. (C. 1885, V.-P. 1902– .) *Trans.* 6.

1902 Legg, Thomas Percy, M.B., 141, Harley Street, Cavendish Square, W.

1893 Lendon, Edwin Harding, M.D., 162, Holland Park Avenue, W.

1899 Leslie, R. Murray, M.D., 26, Harley Street, Cavendish Square, W *C.C.* 1.

1901 Lewers, Arthur Hamilton Nicholson, M.D., 72, Harley Street, Cavendish Square, W.

1892 Lewis, Edward John, M.B., B.C., 74, Hamilton Terrace, N.W.

1895 Lewis, Ernest E., M.D., 30, Weymouth Street, W.

1890 Little, John Fletcher, M.B., 32, Harley Street, W. *C.C.* 5.

1891 Littlewood, Harry, 25, Park Square, Leeds. *Trans.* 1.

1875 Liveing, Edward, M.D., 52, Queen Anne Street, W.

1885 Lockwood, Charles Barrett, 19, Upper Berkeley Street, W. (C. 1896–9.) *Trans.* 7.

Elected

1876 Longhurst, Arthur Edwin Temple, M.D., 26, Lower Belgrave Street, Eaton Square, S.W. (C. 1889–91.) *Trans.* 1.

1897 Low, Harold, M.B., 10, Evelyn Gardens, South Kensington, S.W.

1899 Low, V. Warren, M.D., B.S., 27, Queen Anne Street, Cavendish Square, W.

1881 Lubbock, Montagu, M.D., 19, Grosvenor Street, W.

1876 Lucas, R. Clement, M.B., B.S., 50, Wimpole Street, W. (C. 1883–5, V.P. 1901–3.) *Trans.* 11. *C.C.* 1.

1894 Luff, Arthur Pearson, M.D., 9, Queen Anne Street, Cavendish Square, W.

1879 Lunn, John Reuben, New Marylebone Infirmary, Rackham Street, Ladbroke Grove Road, W. (C. 1890–1.) *Trans.* 8, *C.C.* 17.

1893 Lys, Henry Grabham, M.D., Southbrook, Suffolk Road, Bournemouth.

1889 MacBride, P., M.D., 16, Chester Street, Edinburgh.

1891 MacDonald, Greville, M.D., 85, Harley Street, W.

1901 McGavin, Laurie Hugh, 6, Mansfield Street, Cavendish Square, W. *Trans.* 1, *C.C.* 2.

1881 McHardy, Malcolm Macdonald, 5, Savile Row, W. *Trans.* 1.

1882 Mackenzie, Frederic Morell, 29, Hans Place, S.W.

1891 Mackenzie, Hector W. G., M.A., M.D., 34, Upper Brook Street, W. (C. 1902– .) *Trans.* 6, *C.C.* 1.

1879 Mackenzie, Sir Stephen, M.D., 18, Cavendish Square, and Merrycourt, Gt. Bookham, Leatherhead. (C. 1884, 1888–90, S. 1885–7. V.P. 1900–2.) *Trans.* 9, *C.C.* 10.

1884 Mackern, John, M.B., St. German's Lodge, Shooter's Hill Road, Blackheath, S.E.

1885 Maclaren, Roderick, M.D., Portland Square, Carlisle. *Trans.* 1.

1898 Macready, Jonathan F.C.H., 42, Devonshire Street, Portland Place, W.

1879 Magill, James, M.D., M.C., Coldstream Guards, Queen Anne's Mansions, S.W.

1885 Maguire, Robert, M.D., 4, Seymour Street, W. *Trans.* 1.

1881 Makins, George Henry, C.B. (*Treasurer*), 47, Charles Street, Berkeley Square, W. (C. 1892–4, 1898–1901, S. 1895–8, T. 1903– .) *Trans.* 5, *C.C.* 7.

1887 Malcolm, John D., M.B., C.M., 13, Portman Street, W. (C. 1900–3.) *Trans.* 1, *C.C.* 2.

1890 Manson, Sir Patrick, K.C.M.G., M.D., C.M., F.R.S., 21, Queen Anne Street, W. (C. 1895–7.) *C.C.* 1.

1902 Marriott, Cecil, M B., B.C., 5, Welford Road, Leicester.

1888 Marriott, Hyde, M.B., Dial House, Stockport.

1868 Marsh, Professor F. Howard, 14, Hertford Street, Mayfair, W. (C. 1876–7, 1881–3, S. 1878–80, V.P. 1887–8, T. 1899–1901, P. 1901–3.) *Trans.* 17, *C.C.* 7.

1887 Martin, Sidney, M.D., B.S., F.R.S., 10, Mansfield Street, W. (C. 1896–9.)

1888 Mason, David James, M.D., C.M., Rosemont, Maidenhead.

Elected

1892 Masters, John Alfred, M.D., 31, Albert Gate, Hyde Park, S.W.
1884 Maudsley, Henry Carr, M.D., 22, Collins Street, Melbourne, Victoria.
1897 May, Chichester Gould, M.D., 59, Cadogan Place, S.W.
1868 May, Edward Hooper, M.D., High Cross, Tottenham, Middlesex.
1888 May, William Page, M.D., B.Sc., 9, Manchester Square, W.; and Helouan, nr. Cairo, Egypt (October to April).
1901 Menzies, F. U., Edinburgh University Union, Edinburgh.
1888 Menzies, J. Herbert, 47, Earl's Court Square, S.W.
1893 Mercer, William Bracewell, M.B., B.C., Cliffe Cottage, Ripponden, near Halifax, Yorks.
1878 Meredith, William Appleton, C.M., 21, Manchester Square, W. (C. 1887–9.) *Trans.* 2.
1894 Michels, Ernst, M.D., 48, Finsbury Square, E.C.
1873 Mickle, William Julius, M.D., Grove Hall Asylum, Bow, E. (C. 1897–1900.)
1890 Miley, Miles, M.A., M.B., 21, Belsize Avenue, Hampstead, N.W.
1896 Mills-Roberts, Robert Herbert, Llanberis, North Wales. *Trans.* 1.
1882 Money, Angel, M.D., Hunter Street, Sydney, New South Wales. (C. 1888–90.) *Trans.* 3.
1902 Moon, R. O., M.D., 50, Green Street, Grosvenor Square, W.
1874 Morgan, John Hammond, C.V.O., 68, Grosvenor Street, W. (C. 1883–5, V.P. 1898–1901.) *Trans.* 2, *C.C.* 8.
1888 Morison, Alexander, M.D., 14, Upper Berkeley Street, W. *Trans.* 2, *C.C.* 1.
1899 Morison, Rutherford, 14, Saville Row, Newcastle-on-Tyne. *Trans.* 1.
1877 Morris, Henry, M.A., M.B., 8, Cavendish Square, W. (C. 1884–6, V.P. 1903– .) *Trans.* 9.
1877 Morris, Malcolm Alex., 8, Harley Street, W. (C. 1890–2. *Trans.* 1, *C.C.* 1.
1901 Morse, Thomas Henry, All Saints' Green, Norwich. *Trans.* 1.
1902 Morton, Charles Alfred, 14, Vyvyan Terrace, Clifton, Bristol. *Trans.* 1.
1885 Mott, Frederick Walker, M.D., F.R.S., 25, Nottingham Place, W. (C. 1895–8.) *Trans.* 1, *C.C.* 4.
1899 Mouat-Biggs, Charles E. Forbes, 2, Fauconberg Villas, Bayshill, Cheltenham. *C.C.* 2.
1879 Moullin, Charles W. Mansell, 69, Wimpole Street, W. (C. 1888–90.) *Trans.* 11, *C.C.* 4.
1899 Moynihan, Berkeley George Andrew, M.S., 33, Park Square, Leeds. *Trans.* 1.
1875 Murphy, Shirley F., 9, Bentinck Terrace, Regent's Park, N.W. (C. 1888–90.) *C.C.* 1.
1885 Murray, Alexander Dalton, M.B., Colombo, Ceylon.
1893 Murray, George Redmayne, M.D., 11, Ellison Place, Newcastle-on-Tyne.

Elected

1883 Murray, Hubert Montague, M.D., 25, Manchester Square, W. (C. 1893-6.) *Trans.* 1.

1894 Murray, John, 110, Harley Street, W. *C.C.* 1.

1872 Myrtle, Andrew S., M.D., 10, Park Parade, Harrogate. (C. 1892.)

1902 Nabarro, David, M.D., 6, Crown Office Row, Inner Temple, E.C.

1892 Nash, Walter Gifford, 31, St. Peter's, Bedford.

1889 Newman, D., M.D., 18, Woodside Place, Glasgow. *Trans.* 4.

O.M. Norton, Arthur Trehern, C.B., Leyfields Wood, Ashampstead, Berks. (C. 1874-6.) *Trans.* 7.

1880 O'Connor, Bernard, M.D., 25, Hamilton Road, Ealing, W. *Trans.* 1.

1899 Ogle, Cyril, M.B., 96, Gloucester Place, Portman Square, W. *Trans.* 1.

O.M. Ogle, John William, M.D., 96, Gloucester Place, Portman Square, W. (C. 1867-8, V.P. 1884-5.) *Trans.* 6.

1868 Ogle, William, M.D., The Elms, Driffield Road, Derby.

1887 Oliver, Thomas, M.D., 7, Ellison Place, Newcastle-upon-Tyne.

1887 Openshaw, Thomas Horrocks, M.B., C.M.G., 16, Wimpole Street, W. *Trans.* 1, *C.C.* 1.

1863 Oppert, Franz, M.D., 128, Leipzigerstrasse, Friedenau, Germany. *Trans.* 1.

1889 Ord, W. Wallis, M.D., B.Ch., The Hall, Salisbury. *Trans.* 1, *C.C.* 1.

1887 Ormerod, Joseph Arderne, M.D., 25, Upper Wimpole Street, W. (C. 1899-1902.) *C.C.* 2.

1884 Ormsby, Lambert Hepenstal, M.D., 92, Merrion Square West, Dublin.

1883 Orton, George Hunt, M.B., 1A, Campden Hill Road, Kensington, W.

1877 Owen, Sir Isambard, M.D., 40, Curzon Street, W. (C. 1888-90.) *Trans.* 4.

1888 Oxley, Alfred Rice, M.D., 7, Courtfield Road, South Kensington, S.W.

1888 Page, Frederick, M.D., 1, Saville Place, Newcastle-on-Tyne.

1875 Page, Herbert W., M.C., M.B., 146, Harley Street, W. (C. 1882-4, V.P. 1899-1902.) *Trans.* 8, *C.C.* 2.

1884 Paget, Stephen, 70, Harley Street, W. (C. 1898-1901.) *Trans.* 8, *C.C.* 3.

1881 Parker, Rushton, M.B., B.S., 59, Rodney Street, Liverpool. (C. 1896-9.) *Trans.* 1, *C.C.* 2.

1890 Parkin, Alfred, M.S., 24, Albion Street, Hull. *Trans.* 2.

1894 Parkinson, John Porter, M.D., 57, Wimpole street, W. *Trans.* 1, *C.C.* 4.

1898 Partridge, Arthur A. H., M.D., M.Ch., St. Germans, Sutton, Surrey.

1881 Pasteur, William, M.D. (*Hon. Secretary*), 4, Chandos Street, W. (C. 1891-3, S. 1902- .) *Trans.* 5, *C.C.* 5.

1893 Paterson, Donald Rose, M.D., C.M., 18, Windsor Place, Cardiff.

1900 Paton, Edward Percy, M.S., 84, Park Street, Grosvenor Square, W *Trans.* 1.

Elected

1892 Paul, Frank Thomas, 38, Rodney Street, Liverpool. *Trans.* 1.

1893 Paul, John Ernest, M.B., 26, Queensborough Terrace, Kensington Gardens, W. *Trans.* 1.

O.M. Pavy, Frederick William, M.D., F.R.S., 35, Grosvenor Street, W. (C. 1869–71, V.P. 1882–4.) *Trans.* 3.

1886 Payne, Joseph Frank, M.D., 78, Wimpole Street, W. (C. 1897–1900.) *Trans.* 1.

1902 Pearson, S. Vere, M.B., B.C., 8, Serjeants' Inn, E.C. *C.C.* 2.

1879 Peel, Robert, 120, Collins Street East, Melbourne, Victoria.

1886 Penny, William John, Coombe, West Crewkerne, Somerset.

1887 Penrose, Francis George, M.D., 84, Wimpole Street, W. (C. 1896–9.) *Trans.* 1, *C.C.* 2.

1882 Pepper, Augustus Joseph, M.S., M.B., 13, Wimpole Street, W. *Trans.* 1.

1900 Perkins, George Steele, M.D., 85, Wimpole Street, W.

1898 Perkins, J. J., M.D., 41, Wimpole Street, Cavendish Square, W. *Trans.* 1, *C.C.* 1.

1895 Phear, Arthur G., M.D., B.C., 47, Weymouth Street, Portland Place, W. *Trans.* 2.

1895 Phillips, F. B. Willmer, M.B., 7, Harper Place, Bedford.

1884 Phillips, Sidney, M.D., 62, Upper Berkeley Street, W. (C. 1893–6.) *Trans.* 7, *C.C.* 6.

O.M. Pick, Thomas Pickering, The Nook, Great Bookham, Surrey. (S. 1874–7, C. 1878–80, V.P. 1885–6.) *Trans.* 4.

1885 Pitt, George Newton, M.D., 15, Portland Place, W. (C. 1894–6.) *Trans.* 6, *C.C.* 5.

1883 Pitts, Bernard, M.A., M.C., 109, Harley Street, W. (C. 1893.) *Trans.* 5.

1871 Playne, Alfred, M.B., Maidenhead.

1884 Poland, John, 2, Mansfield Street, Cavendish Square, W.

1884 Pollard, Bilton, B.S., 24, Harley Street, W. (C. 1895–8.) *Trans.* 2.

1868 Pollock, James Edward, M.D., 37, Collingham Place, S.W. (C. 1878–80.)

1871 Poore, George Vivian, M.D., 24a, Portland Place, W. (C. 1879–81, V. P. 1901–3.) *Trans.* 4.

1881 Powell, H. A., M.A., 44, Sandgate Road, Folkestone.

O.M. Powell, Sir R. Douglas, Bart., K.C.V.O., M.D., 62, Wimpole Street, W. (C. 1874–6, V.P. 1889–90, P. 1899-1901.) *Trans.* 4.

1868 Prentis, Charles, Surgeon-Major, Bengal Medical Service; India.

1902 Price, F. W., M.B., 1, Maida Vale Mansions, W.

1884 Pringle, John James, M.B., 23, Lower Seymour Street, W. (C. 1890–2, 1897–1900.) *Trans.* 1, *C.C.* 3.

1884 Pye-Smith, Philip Henry, M.D., F.R.S., 48, Brook Street, W. (C. 1890–2.) *Trans.* 1.

ELECTED

1896 PYE-SMITH, RUTHERFORD JOHN, 350, Glossop Road, Sheffield.

1893 RAKE, ALFRED THEODORE, M.B., B.S., 58, Delancey Street, Regent's Park, N.W.

1895 RAMSAY, HERBERT MURRAY, 35A, Hertford Street, Mayfair, W.

1889 RANKING, JOHN E., M.D., Hanover House, Tunbridge Wells.

1883 READ, THOMAS LAURENCE, 11, Petersham Terrace, Queen's Gate, S.W.

1902 REID, Sir JAMES, Bart., G.C.V.O., K.C.B., 72, Grosvenor Street, Grosvenor Square, W.

1901 REISSMANN, CHARLES, M.D., Knutsford, Glenelg, South Australia.

1889 **Rennie**, GEORGE EDWARD, M.D., College Street, Hyde Park, Sydney, N.S.W.

1868 RICE, MICHAEL W., M.D., Elmbank, Hargrave, Northamptonshire, (C. 1876–8.)

1898 RICHARDS, JOSEPH STEWART, M.D., B.S., The Infirmary, East Dulwich, S.E. *C.C.* 2.

O.M. RINGER, SYDNEY, M.D., F.R.S., 15, Cavendish Place, W. (C. 1871–2.) *Trans.* 1.

1896 RISDON, WILLIAM ELLIOT, M.D., "Oakfield," Woodside Park Road, North Finchley, N.

1902 RIVIERE, CLIVE, M.D., 19, Devonshire Street, W. *C.C.* 1.

1873 **Roberts**, DAVID LLOYD, M.D., 11, St. John Street, Manchester.

1888 ROBERTS, FRANK ERNEST, Tulse Dale Villa, Lower Norwood, S.E.

1883 ROBERTS, FREDERICK THOMAS, M.D., 102, Harley Street, W. (C. 1892–4.)

1900 ROBERTS, GRIFFITH LLOYD, M.B., C.M., Castle Square, Carnarvon, North Wales.

1890 ROBERTSON, ROBERT, M.D., Belgrave Road, Ventnor, Isle of Wight.

1885 ROBINSON, ARTHUR HENRY, M.D., St. Mary's Infirmary, Highgate Hill, N. *C.C.* 3.

1890 ROBINSON, GEORGE SOMERVILLE, Surgeon-Major, 13, Lupus Street, St. George's Square, S.W.

1892 ROBINSON, HENRY BETHAM, M.D., M.S., 1, Upper Wimpole Street, W. (C. 1901–). *Trans.* 7, *C.C.* 10.

1885 ROBSON, A. W. MAYO, 8, Park Crescent, W., and 7, Park Square, Leeds. (C. 1893–6.) *Trans.* 15.

1897 ROGER-SMITH, HUGH, M.D., 1, College Terrace, Fitzjohn's Avenue, N.W. *Trans.* 1.

1889 ROLLESTON, HUMPHRY DAVY, M.A., M.D., 55, Upper Brook Street, Grosvenor Square, W. (C. 1899–1902.) *Trans.* 10. *C.C.* 9.

1897 ROSS, FREDERICK WILLIAM FORBES, M.D., 15, Gower Street, W.C.

1877 **ROTH**, BERNARD, 38, Harley Street, Cavendish Square, and "Wayside,' Preston Park Avenue, Brighton. *Trans.* 1, *C.C.* 4.

1890 ROUGHTON, EDMUND WILKINSON, B.S., 38, Queen Anne Street, W. *Trans.* 1, *C.C.* 1.

1874 ROWLAND, EDWARD ROGER, Dordrecht, Wodehouse, S. Africa.

Elected

1898 Russell, Alfred Ernest, M.D., B.S., 107, Lambeth Palace Road, Albert Embankment, S.E. *C.C.* 1.

1900 Russell, J. S. Risien, M.D., 44, Wimpole Street, Cavendish Square, W. *Trans.* 1, *C.C.* 1.

1887 Rutherfoord, H. T., M.B., Salisbury House, Taunton.

1885 Ryle, Reginald John, M.D., 15, German Place, Brighton.

1882 Sainsbury, Harrington, M.D., 52, Wimpole Street, W. (C. 1898–1901.)

1899 Sandifer, Henry Stephen, M.D., B.S., 11, Porchester Gardens, W.

1893 Sansom, Arthur Ernest, M.D., 84, Harley Street, W. *Trans.* 1, *C.C.* 1.

1873 Savage, George Henry, M.D., 3, Henrietta Street, W. (C. 1882–3.)

1886 Savill, Thomas Dixon, M.D., 60, Upper Berkeley Street, W. *Trans.* 1, *C.C.* 5.

1899 Schorstein, Gustave, M.B., 11, Portland Place, W.

1886 Scott, Alfred, 15, German Place, Brighton.

1894 Scott, Bernard, "Hartington," Poole Road, Bournemouth.

1900 Scott, Kenneth, M.D., 7, Manchester Square, W.

1892 Scott, Richard James Herbert, 28, Circus, Bath. *C.C.* 1.

1902 Sears, Alfred Ernest, 33, Lee Terrace, Blackheath, S.E. *Trans.* 1.

1869 Sedgwick, Leonard William, M.D., 69, Gloucester Terrace, Hyde Park, W. (C. 1879–81, V.P. 1897–1900.)

1897 Segundo, Charles Sempill de, M.B., B.S., 6, Brook Street, Hanover Square, W.

1892 Selwyn-Harvey, John Stephenson, M.D., 1, Astwood Road, S.W.

1878 Semon, Sir Felix, C.V.O., M.D., 39, Wimpole Street, W. (C. 1885–7.) *Trans.* 8, *C.C.* 3.

1884 Sharkey, Seymour J., M.D., 22, Harley Street, W. (C. 1895–8.)

1898 Shaw, Harold Batty, M.D., 7, Devonshire Street, Portland Place, W. *C.C.* 4.

1889 Shaw, Lauriston Elgie, M.D., 64, Harley Street, W. *C.C.* 1.

1875 Sherwood, Arthur Paul, 8, Seaside Road, Eastbourne.

1886 Silcock, Arthur Quarry, M.D., B.S., 52, Harley Street, W. (C. 1896–9.) *Trans.* 5. *C.C.* 2.

1903 Simpson, Graham, 1, Bartlett's Passage, Holborn Circus, E.C. *C.C.* 1.

1879 Skerritt, Edward Markham, M.D., Edgecumbe House, Richmond Hill, Clifton, Bristol. (C. 1895–8.) *Trans.* 2.

1872 Slight, George, M.D., 14, Old Burlington Street, W.

1882 Smith, E. Noble, 24, Queen Anne Street, W. *Trans.* 1.

1896 Smith, Ebenezer Stanley, M.D., 68, Wimpole Street, Cavendish Square, W.

1888 Smith, Frederick J., M.D., 138, Harley Street, W. *Trans.* 2.

1884 Smith, R. Percy, M.D., 36, Queen Anne Street, Cavendish Square, W. (C. 1898–1901.)

O.M. Smith, Sir Thomas, Bart., 5, Stratford Place, W. (C. 1869–71, V.P. 1880–2.) *Trans.* 14.

ELECTED

1894 SMITH, THOMAS RUDOLPH, M.B., B.C., 25, Bridge Road, Stockton-on-Tees.

1893 SNAPE, ERNEST ALFRED, M.D., 41, Welbeck Street, W.

1868 SNOW, WILLIAM V., M.D., Richmond Gardens, Bournemouth.

1890 SOLLY, ERNEST, M.B., Strathlea, Harrogate, Yorks. *C.C.* 1.

1888 SPENCER, WALTER GEORGE, M.S., M.B., 35, Brook Street, W. (C. 1896–9.) *Trans.* 11, *C.C.* 11.

1885 SPICER, FREDERICK, M.D., 17, Wimpole Street, Cavendish Square, W.

1888 SPICER, ROBERT HENRY SCANES, M.D., 28, Welbeck Street, W. *C.C.* 1.

1882 SPOONER, FREDERICK HENRY, M.D., 4, Maitland Place, Lower Clapton, N.E.

1896 SPURRELL, CHARLES, Medical Superintendent, Poplar and Stepney Sick Asylum, Devon's Road, Bromley-by-Bow, E.

1876 SQUIRE, A. BALMANNO, 24, Weymouth Street, W. *Trans.* 5, *C.C.* 4.

1892 STABB, EWEN CARTHEW, 57, Queen Anne Street, W. *C.C.* 1.

1879 STAPLES, FRANCIS PATRICK, Brigade-Surgeon, Army (retired). (Address uncommunicated.)

1896 STEWARD, FRANCIS JAMES, M.B., B.S., 133, Harley Street, W. *Trans.* 2.

1874 **Stirling**, EDWARD C., M.D. [care of Messrs. ELDER & Co., 7, St. Helen's Place, E.C.], Adelaide, South Australia.

1888 STOKER, GEORGE, 14, Hertford Street, W. *C.C.* 2.

1899 STONE, WILLIAM GREAM, M.B., 93, Denmark Hill, S.E.

1884 STONHAM, CHARLES, C.M.G., 4, Harley Street, W. *C.C.* 3.

1878 STRUGNELL, FREDERICK WILLIAM, Grove End House, Highgate Road, Highgate, N.W. (C. 1903– .) *C.C.* 1.

1878 **Sturge**, WILLIAM ALLEN, M.D., 29, Boulevard Dubouchage, Nice, France.

1894 SUTHERLAND, GEORGE A., M.D., 73, Wimpole Street, Cavendish Square, W. *Trans.* 1.

1882 SYMONDS, CHARTERS JAMES, M.S., 58, Portland Place, W. (C. 1888–9, 1901– , S. 1898–1901.) *Trans.* 12, *C.C.* 17.

1876 SYMONDS, HORATIO PERCY, 35, Beaumont Street, Oxford.

1885 TAIT, EDWARD SABINE, M.D., 48, Highbury Park, N.

1885 TAIT, HENRY BREWER, Lincludeu, Sunnyside Road, Hornsey Lane, N.

1891 TATE, WALTER WILLIAM HUNT, M.D., 32, Queen Anne Street, W.

1886 TAY, WAREN, 4, Finsbury Square, E.C.

1878 TAYLER, FRANCIS THOMAS, M.B., 224, Lewisham High Road, S.E.

1897 TAYLOR, E. CLAUDE, M.D., B.S., Eland House, Rosslyn Hill, Hampstead, N.W.

1875 **TAYLOR**, FREDERICK, M.D., (*President*), 20, Wimpole Street, W. (S. 1879–81, C. 1882–4, V.P. 1894–7, T. 1901–3, P. 1903– .) *Trans.* 13, *C.C.* 3.

ELECTED

1889 **Taylor**, HENRY HERBERT, 10, Brunswick Place, Hove, Sussex.
1890 TAYLOR, JAMES, M.D., 49, Welbeck Street, W. *C.C.* 3.
1882 TAYLOR, SEYMOUR, M.D., 16, Seymour Street, W. *Trans.* 1, *C.C.* 4.
1885 **Taylor**, W. C. EVERLEY, 34, Queen Street, Scarborough.
1886 TEALE, THOMAS PRIDGIN, M.B., F.R.S., 38, Cookridge Street, Leeds. (C. 1897–1900.)
1896 TEMPLETON, GEORGE, M.B., 10, Seymour Street, Portman Square, W.
1890 THANE, EDGAR HERBERT, M.D., Wagga-Wagga, New South Wales.
1882 THIN, GEORGE, M.D., 63, Harley Street, W. *Trans.* 1.
1886 THOMPSON, CHARLES HERBERT, M.D., 133, Harley Street, Cavendish Square, W.
O.M. THOMPSON, EDMUND SYMES, M.D., 33, Cavendish Square, W. (C. 1880–2.) *Trans.* 1.
O.M. THOMPSON, Sir HENRY, Bart., 35, Wimpole Street, W. (C. 1867–8, V.P. 1886–7.) *Trans.* 3.
1897 THOMSON, H. CAMPBELL, M.D., 34, Queen Anne Street, Cavendish Square, W. *C.C.* 1.
1894 THOMSON, STCLAIR, M.D., 28, Queen Anne Street, W. *Trans.* 2, *C.C.* 8.
1896 THORNE, WILLIAM BEZLY, M.D., 53, Upper Brook Street, W. *C.C.* 2.
1887 THORNTON, JOHN KNOWSLEY, M.B., C.M., Hildersham Hall, Cambridge. (C. 1890–1.)
1872 THORNTON, WILLIAM PUGIN, 35, St. George's Road, Canterbury. *Trans.* 5.
1885 THURSFIELD, THOMAS WILLIAM, M.D., Selwood, Beauchamp Square, Leamington.
1899 TILLEY, HERBERT, M.D., 89, Harley Street, Cavendish Square, W. *C.C.* 1.
1891 TOMSON, W. BOLTON, M.D., Park Street West, Luton, Bedfordshire.
1897 TOOGOOD, F. SHERMAN, M.D., The Infirmary, 282, High Street, Lewisham, S.E.
1892 TOOTH, HOWARD HENRY, C.M.G., M.D., 34, Harley Street, W. *Trans.* 1.
1887 TOTSUKA, KANKAI, Tokio, Japan.
1874 TRAVERS, WILLIAM, M.D., 2, Phillimore Gardens, Kensington, W.
1884 TREVES, Sir FREDERICK, Bart., C.B., K.C.V.O., 6, Wimpole Street, W. (C. 1893.) *Trans.* 7.
1897 TUBBY, ALFRED HERBERT, M.S., 25, Weymouth Street, Portland Place, W. *Trans.* 2, *C.C.* 3.
1899 TULLIS, GEORGE AUGUSTUS, M.D., Holmbush, Grove Road, Southsea.
1882 TURNER, GEORGE ROBERTSON, 41, Half Moon Street, W. (C. 1897–1900.) *Trans.* 9. *C.C.* 6.
1888 TURNER, PHILIP DYMOCK, M.D., Sudbury, Isle of Wight. *Trans.* 1.
1898 TURNER, WILLIAM, M.B., 53, Queen Anne Street, Cavendish Square, W.
1893 TURNEY, HORACE GEORGE, M.D., 68, Portland Place, W. *C.C.* 4.
1878 TYSON, WILLIAM JOSEPH, M.D., 10, Langhorne Gardens, Folkestone. (C. 1886–8, V.-P. 1900–2.) *Trans.* 8.

ELECTED

1881 URTHOFF, JOHN CALDWELL, M.D., Wavertree House, Brunswick Place, Brighton. (C. 1898–1901.)

1868 VENNING, EDGCOMBE, 30, Cadogan Place, S.W. (C. 1876–8.) *Trans.* 2.

1890 VOELCKER, ARTHUR FRANCIS, M.D., B.S., 101, Harley Street, W. (C. 1900–3.) *Trans.* 2, *C.C.* 2.

1886 WADE, CHARLES H., 19, Royal Terrace, Southend-on-Sea.

1868 WAGSTAFFE, WILLIAM WARWICK, Purleigh, St. John's Hill, Sevenoaks. (C. 1878.)

1886 **WAINEWRIGHT**, BENJAMIN, M.B., C.M. (address uncommunicated).

1885 WAKLEY, THOMAS, jun., 5, Queen's Gate, S.W.

1899 WALKER, HENRY ROE, 8, Harley Street, W.

1896 WALLACE, CUTHBERT SYDNEY, M.B., 26, Upper Wimpole Street, W. (C. 1902– .) *Trans.* 5, *C.C.* 5.

1890 WALLIS, FREDERICK CHARLES, M.B., B.C., 107, Harley Street, W. (C. 1902– .) *Trans.* 4, *C.C.* 10.

1888 WALTERS, FREDERICK RUFENACHT, M.D., Crooksbury Sanatorium, Farnham, Surrey. *C.C.* 2.

1896 WARD, EDWARD, M.B., 22, Park Place, Leeds. *Trans.* 1.

1888 Warner, PERCY, Rydal, Woodford Green, Essex.

1891 WATERHOUSE, HERBERT FURNIVALL, M.D., C.M., 81, Wimpole Street, W.

1902 WATSON, CHARLES GORDON, 44, Welbeck Street, Cavendish Square, W.

1896 **WEBER**, FRED. PARKES, M.D., 19, Harley Street, W. *Trans.* 1. *C.C.* 7.

O.M. WEBER, Sir HERMANN D., M.D., 10, Grosvenor Street, W. (C. 1867–71, V.P. 1873–5.) *Trans.* 9.

1882 WEST, SAMUEL, M.D., 15, Wimpole Street, W. (C. 1887–9.) *Trans.* 16, *C.C.* 5.

1895 WETHERED, FRANK J., M.D., 83, Harley Street, W.

1874 WHEELHOUSE, CLAUDIUS GALEN, Cliffe Point, Filey, Yorks. *Trans.* 1.

1868 WHIPHAM, THOMAS TILLYER, M.D., 11, Grosvenor Street, W. (C. 1878–80, V.P. 1894–7.) *Trans.* 13.

1891 WHITE, CHARLES PERCIVAL, M.B., B.C., 22, Cadogan Gardens, S.W.

1882 WHITE, EDWIN FRANCIS, Westlands, 280, Upper Richmond Road, Putney, S.W.

1890 WHITE, GILBERT B. MOWER, M.B., B.S., 112, Harley Street, W.

1883 WHITE, WILLIAM HALE, M.D. 65, Harley Street, W. (C. 1887–9, 1896–9, S. June, 1893–6.) *Trans.* 16, *C.C.* 4.

1883 WHITE, WILLIAM HENRY, M.D., 43, Weymouth Street, W. *Trans.* 1. *C.C.* 1.

1894 WHITELOCKE, RICHARD HENRY ANGLIN, M.B., C.M., 6, Banbury Road, Oxford.

1897 WHITFIELD, ARTHUR, M.D., 21, Bentinck Street, Manchester Square, W.

1897 WHITING, ARTHUR JOHN, M.D., 142, Harley Street, W.

1882 WHITTLE, EDWARD GEORGE, M.D., 9, Regency Square, Brighton.

Elected

1871 Wight, George, M.B., C.M., 428, Liverpool Road, N.

1879 Wilcox, Henry, M.B., Newlyn, Fleet, Hants.

1894 **WILKIN**, Griffith Charles, Blakesley, near Towcester, Northamptonshire.

1884 Willcocks, Frederick, M.D., 14, Mandeville Place, W. *C.C.* 1.

O.M. Willett, Alfred, 36, Wimpole Street, W. (C. 1872–5, V.P. 1889–91.) *C.C.* 2.

1890 Willett, Edgar, M.B., 22, Queen Anne Street, Cavendish Square, W.

1888 Williams, Campbell, 18, Queen Anne Street, Cavendish Square, W. *Trans.* 2.

O.M. Williams, Charles Theodore, M.D., 2, Upper Brook Street, W. (C. 1877–9, V.P. 1889–90.) *Trans.* 8.

1888 Williams, Dawson, M.D., 2, Wyndham Place, Bryanston Square, W. (C. 1893–7.)

1903 Williams, Leonard, M.D., 8, York Street, Portman Square, W.

1890 Williams, W. Roger, Beaufort House, Clifton Down, Clifton, Bristol.

O.M. Willis, Francis, M.D., The Spa, Braceborough, Stamford.

1893 Wills, Joseph Pearce Budgett, M.D., Bexhill, Hastings.

1889 Wills, William Alfred, M.D., 29, Lower Seymour Street, W.

1886 Wilson, Albert, M.D., Minto House, South Woodford, Essex. *Trans.* 1, *C.C.* 2.

1888 Wilson, Claude, M.D., C.M., Belmont, Tunbridge Wells. *Trans.* 2.

1891 Wilson, Theodore Stacey, M.D., C.M., 27, Wheeley's Road, Edgbaston, Birmingham. *Trans.* 2.

1890 Wood, Neville, 42, Elvaston Place, Queen's Gate, S.W.

1883 Woodcock, John Rostron, Darlington Court, North Road, Bath.

1879 Woodward, George P. M., M.D., Deputy Surgeon-General; Sydney, New South Wales.

1894 Woollett, Charles Jerome, "Ambleside," Streatham, S.W.

1884 Worts, Edwin, 6, Trinity Street, Colchester.

1892 Wynter, Walter Essex, M.D., B.S., 30, Upper Berkeley Street, W. *C.C.* 4.

1872 Yeo, I. Burney, M.D., 44, Hertford Street, W. (C. 1881–3.) *Trans.* 8.

[*It is requested that any change of Title or Residence be communicated to the Secretaries before the 1st of July in each year, in order that the list may be made as correct as possible.*]

LIST OF MEMBERS.

ORIGINAL MEMBERS (ALPHABETICALLY).

Henry Arnott.
Richard Barwell.
Henry Charlton Bastian, M.D., F.R.S.
Sir Wm. Henry Broadbent, Bart., K.C.V.O., M.D, F.R.S.
Thomas Bryant.
Thomas Buzzard, M.D.
William Cayley, M.D.
Sir William Selby Church, Bart., K.C.B., M.D.
Edward Clapton, M.D.
John Couper.
William Howship Dickinson, M.D.
Sir Dyce Duckworth, M.D.
Christopher Heath.
Timothy Holmes.
Jonathan Hutchinson, F.R.S.
J. Hughlings Jackson, M.D., F.R.S.
Charles Kelly, M.D.
John Langton.
George Lawson.
Arthur Trehern Norton, C.B.
John William Ogle, M.D.
Frederick William Pavy, M.D., F.R.S.
Thomas Pickering Pick.
Sir Richard Douglas Powell, Bart., M.D., K.C.V.O.
Sydney Ringer, M.D., F.R.S.
Sir Thomas Smith, Bart.
Edmund Symes Thompson, M.D.
Sir Henry Thompson, Bart.
Sir Hermann D. Weber, M.D.
Alfred Willett.
Charles Theodore Williams, M.D.
Francis Willis, M.D.

ARRANGED ACCORDING TO DATE OF ELECTION.

1868 Constantine Holman, M.D.
Thomas Tillyer Whipham, M.D.
Christian G. H. Bäumler, M.D.
James Grey Glover, M.D.
T. Henry Green, M.D.
Howard Marsh.
Charles Prentis.
Edgcombe Venning.

1868 John Ford Anderson, M.D.
George Granville Bantock, M.D.
George Charles Bright, M.D.
Frank W. Cooper.
Julian Evans, M.B.
Edward Hooper May, M.D.
William Warwick Wagstaffe.
William Ogle, M.D.

1868 James Edward Pollock, M.D.
Franz Oppert, M.D.
William V. Snow, M.D.
Charles Drage, M.D.
Frederick Royston Fairbank, M.D.
Michael W. Rice, M.D.

1869 Leonard William Sedgwick, M.D.
J. Warrington Haward.
Henry Frederick Augustus Goodridge, M.D.
Olliver Thomas Duke, M.B.

1871 Alfred Playne, M.B.
George Wight, M.B.
Ebenezer Diver, M.D.
George Vivian Poore, M.D.

1872 I. Burney Yeo, M.D.
Henry Harris, M.D.
William Pugin Thornton.
Sir G. Anderson Critchett.
J. C. J. Fenwick, M.D.
Andrew J. Myrtle, M.D.
Sir William Bartlett Dalby.
Thomas Ridge Jones, M.D.
George Slight, M.D.

1873 William Julius Mickle, M.D.
David Lloyd Roberts, M.D.
George Henry Savage, M.D.
Edwin Chisholm, M.D.
Thomas Churton, M.D.

1874 John Hammond Morgan, C.V.O.
Edward R. Rowland.
Claudius Galen Wheelhouse.
Edward C. Stirling, M.D.
Sir William Henry Bennett, K.C.V.O.
William Travers, M.D.
William Brown Holderness.
Andrew Clark.

1875 Sir Thomas Barlow, Bart., K.C.V.O., M.D.
Sidney Coupland, M.D.
Clinton T. Dent.
C. D. B. Hale.
Edward Liveing, M.D.
Rickman John Godlee, M.S.
Arthur Paul Sherwood.
T. Gilbart-Smith, M.D.
James Frederic Goodhart, M.D.
Sir William Richard Gowers, M.D., F.R.S.
William Smith Greenfield, M.D.
Shirley F. Murphy.

1875 Herbert W. Page.
Frederick Taylor, M.D.

1876 Arthur E. J. Barker.
Horatio Percy Symonds.
A. Balmanno Squire.
David White Finlay, M.D.
Sir Henry Greenway Howse, M.S.
Furneaux Jordan.
R. Clement Lucas, B.S.
George Buckston Browne.
Arthur Edwin Temple Longhurst, M.D.

1877 Robert Hogarth Clay, M.D.
A. Pearce Gould, M.S.
Henry Radcliffe Crocker, M.D.
David B. Lees, M.D.
Walter Hamilton Acland Jacobson, M.B., M.Ch.
Sir Isambard Owen, M.D.
William Ewart, M.D.
Henry Morris, M.B.
Henry Ambrose Lediard, M.D.
Bernard Roth.
Henry Hugh Clutton.
Malcolm Alex. Morris.

1878 George P. Field.
Thomas Warner Lacey.
Thomas Colcott Fox, M.B.
Sir Felix Semon, C.V.O., M.D.
Henry de Fonmartin, M.D.
C. H. Golding-Bird, M.B.
Donald William Charles Hood, M.D.
Lord Lister, P.C., O.M., F.R.S.
Francis Thomas Tayler, M.B.
F. de Havilland Hall, M.D.
William Allen Sturge, M.D.
William Joseph Tyson, M.D.
Charles Robert Bell Keetley.
William Appleton Meredith, C.M.

1879 Frederick William Strugnell.
William Edward Burton.
James Magill, M.D.
Edward Markham Skerritt, M.D.
Henry Wilcox, M.B.
John Abercrombie, M.D.
Sir Stephen Mackenzie, M.D.
William Harrison Cripps.
Francis Patrick Staples.
Geo. Courtenay Henderson, M.D.
Henry Davy.
Thos. Walter Harropp Garstang.

1879 Charles W. Mansell Moullin.
John Reuben Lunn.
George P. M. Woodward, M.D.
Robert Peel.
Frederic S. Dennis, M.D.
1880 T. Mark Hovell.
Wyndham Cottle, M.D.
Henry Francis Baker.
Bernard O'Connor, M.D.
Charles Edward Beevor, M.D.
1881 George Henry Makins, C.B.
Robert William Burnet, M.D.
James Kingston Fowler, M.D.
Charles Edward Harrison, M.B.
Malcolm Macdonald McHardy.
Rushton Parker.
Montagu Lubbock, M.D.
William Pasteur, M.D.
John Caldwell Uhthoff, M.D.
Henry Trentham Butlin.
H. A. Powell, M.A.
1882 George Robertson Turner.
E. Noble Smith.
Robert William Goldie.
Frederick Charles Barker, M.D.
Frederic Morell Mackenzie.
Daniel Colquhoun, M.D.
Seymour Taylor, M.D.
Philip Henry Bindley, M.B.
Edward George Whittle, M.D.
D. H. Goodsall.
Frederick Henry Spooner, M.D.
Frederick Haycraft Berry, M.D.
Herbert Collier, M.D.
Samuel West, M.D.
Emile Emond, M.D.
Charters James Symonds, M.S.
Angel Money, M.D.
C. F. Coxwell, M.B.
Augustus Joseph Pepper, M.B.
Harrington Sainsbury, M.D.
George Thin, M.D.
Edwin Francis White.
1883 Anthony A. Bowlby, C.M.G.
Percy Kidd, M.D.
William Henry White, M.D.
Hubert Montague Murray, M.D.
Robert Fitzroy Benham.
William Henry Allchin, M.D.
John Mitchell Bruce, M.D.
William Arbuthnot Lane, M.S.
Bernard Pitts.
William Hale White, M.D.
William Coode Adams, M.B.
1883 Robert Leamon Bowles, M.D.
George Henry Jackson.
George Hunt Orton, M.B.
Thomas Laurence Read.
Frederick Thomas Roberts, M.D.
Charles Alfred Ballance, M.S.
John Rostron Woodcock.
1884 Frederick Willcocks, M.D.
R. Percy Smith, M.D.
Edgar Duke.
John Mackern, M.B.
Paul M. Chapman, M.D.
Wilmot Parker Herringham, M.D.
Philip Henry Pye-Smith, M.D. F.R.S.
Charles Stonham, C.M.G.
Dudley Wilmot Buxton, M.D.
Edwin Worts.
Seymour J. Sharkey, M.D.
Sir Frederick Treves, Bart., C.B., K.C.V.O.
John James Pringle, M.B.
Frederick Lucas Benham, M.D.
Walter Edmunds, M.C.
Stephen Paget.
Lambert Hepenstal Ormsby, M.D.
John Poland.
Edwin Leonard Adeney, M.D.
Sir Victor Horsley, F.R.S.
Henry Carr Maudsley, M.D.
Bilton Pollard, B.S.
1885 Frederick Spicer, M.B.
Herbert Larder.
James Berry, B.S.
Fred. Walker Mott, M.D., F.R.S.
George Newton Pitt, M.D.
W. C. Everley Taylor.
Sidney Phillips, M.D.
A. W. Mayo Robson.
Thomas Wakley, jun.
Herbert William Allingham.
Thomas William Thursfield, M.D.
Alexander Dalton Murray, M.B.
Robert Maguire, M.D.
Robert Alexander Gibbons, M.D.
Henry Brewer Tait.
Richard Caton, M.D.
Arthur Henry Robinson, M.D.
Edward Sabine Tait, M.B.
William Bruce Clarke, M.B.
Charles Barrett Lockwood.
Reginald J. Ryle, M.D.
J. Michell Clarke, M.D.

1885 Henry George Armstrong.
Roderick Maclaren, M.D.
W. Watson Cheyne, C.B., F.R.S.
Edward Liveing Fenn, M.D.
1886 Thomas Dixon Savill, M.D.
Charles Henry Wade.
Benjamin Wainewright, M.B.
Waren Tay.
William John Penny.
William Henry Battle.
James Hardie, M.D.
Francis Henry Hawkins, M.D.
R. Hingston Fox, M.D.
John Ward Cousins, M.D.
Joseph Frank Payne, M.D.
T. Pridgin Teale, F.R.S.
H. Lankester, M.D.
Arthur T. Davies, M.D.
Charles Herbert Thompson, M.D.
Arthur Quarry Silcock, M.D., B.S.
Henry Handford, M.D.
Alfred Scott.
Albert Wilson, M.D.
1887 Archibald E. Garrod, M.D.
H. T. Rutherfoord, M.B.
Kankai Totsuka.
Thomas Oliver, M.D.
Francis George Penrose, M.D.
Samuel Herbert Habershon, M.D.
John Knowsley Thornton, M.B., C.M.
Oswald Auchinleck Browne, M.D.
Joseph Arderne Ormerod, M.D.
J. H. E. Brock, M.B., B.S.
Francis William Clark.
A. H. Weiss Clemow, M.D., C.M.
E. Hurry Fenwick.
R. Lawford Knaggs, B.C.
John D. Malcolm, M.B., C.M.
Sidney Martin, M.D., B.S., F.R.S.
Thomas Horrocks Openshaw, C.M.G., M.B.
1888 A. G. Barrs, M.D.
J. W. Batterham, M.B., B.S.
Montagu Handfield-Jones, M.D.
Alfred Rice Oxley, M.D.
Robert Henry Scanes Spicer, M.D.
Campbell Williams.
Frederic S. Eve.
Alexander Morison, M.D.
Frederick Page, M.D.
Frederick J. Smith, M.D.
Frederick R. Walters, M.D.
Claude Wilson, M.D., C.M.
1888 Charles H. Gage-Brown, M.D.
J. H. Menzies.
Frank Ernest Roberts.
George Stoker.
Robert Ashton Bostock.
Hugh Armstrong.
Hyde Marriott, M.B.
Percy Warner.
J. T. James, M.D.
Edwin A. Barton.
W. Page May, M.D.
Philip D. Turner, M.D.
Dawson Williams, M.D.
Augustus W. Addinsell, M.B., C.M.
John Anderson, M.D.
Henry French Banham, M.D.
David James Mason, M.D., C.M.
Walter G. Spencer, M.B., M.S.
1889 Theodore Dyke Acland, M.D.
Raymond Johnson, M.B., B.S.
H. D. Rolleston, M.A., M.D.
P. MacBride, M.D.
D. Newman, M.D.
Herbert Elwin Harris, M.B.
John E. Ranking, M.D.
William Alfred Wills, M.D.
Edward Ashby Fardon.
Stanley Boyd, B.S.
George Ezra Halstead, M.D., B.S.
Henry Herbert Taylor.
John Duncan, M.D.
Wm. Wallis Ord, M.D., B.Ch.
Leonard Arthur Bidwell.
Arthur J. M. Bentley, M.D.
Francis R. B. Bisshopp, M.B.
Henry Percy Dean, M.B.
Louis Albert Dunn, M.S.
Daniel Mackay Forbes.
H. Pennell Hawkins, M.D., B.Ch.
Lauriston Elgie Shaw, M.D.
1890 John Rose Bradford, M.D., F.R.S.
J. Fletcher Little, M.B.
Robert Robertson, M.D.
Ernest Solly, M.B.
James Taylor, M.D.
Francis O. Buckland, B.A., M.B., C.M.
G. Somerville Robinson.
Edmund W. Roughton, B.S.
Edgar Willett, M.B.

1890 James Calvert, M.D.
H. Roxburgh Fuller, M.D.
Arthur F. Voelcker, M.D.
Neville Wood.
W. Roger Williams.
Gilbert B. M. White, M.B., B.S.
Frederick Charles Wallis, M.B., B.C.
Alfred Parkin, M.S.
George A. Hawkins-Ambler.
James Harper, M.D.
Walter Henry Brown.
John Walter Carr, M.D.
Ernest Le Cronier Lancaster, M.B., B.Ch.
Sir Patrick Manson, K.C.M.G., M.D., F.R.S.
Miles Miley, M.A., M.B.
Edgar Herbert Thane, M.B.
Charles William Chapman, M.D.
Michael G. Foster, M.A., M.D.

1891 Frederic François Burghard, M.D., M.S.
Roger Neville Goodman, M.B.
Herbert Furnivall Waterhouse, M.D., C.M.
Walter William Hunt Tate, M.D.
William Lee Dickinson, M.D.
Greville MacDonald, M.D.
J. Kingston Barton.
J. Dundas Grant, M.D.
W. Kington Fyffe, M.B., B.C.
Albert Carless, M.B., M.S.
W. Bolton Tomson, M.D.
Harry Littlewood, M.D.
Hector W. G. Mackenzie, M.A., M.D.
Chas. Percival White, M.B., B.C.
Arnold Caddy.
Theodore Stacey Wilson, M.D., C.M.

1892 William Hunter, M.D.
Frank Thomas Paul.
Walter Essex Wynter, M.D., B.S.
Damer Harrison.
John Alfred Masters, M.D.
Walter Giffard Nash.
John Stephenson Selwyn-Harvey, M.D.
Ewen Carthew Stabb.
Edward John Lewis, M.B., B.C.
Henry Betham Robinson, M.D. M.S.

1892 Richard James Herbert Scott.
Howard Henry Tooth, C.M.G., M.D.

1893 John Ernest Paul, M.B.
James William Bond, M.D.
Harry Campbell, M.D.
W. Soltau Fenwick, M.D.
Ernest Alfred Snape, M.D.
Lewis G. Glover, M.B., B.C.
William Bracewell Mercer, M.B., B.C.
Robert Henry Cole, M.D.
Donald Rose Paterson, M.D., C.M.
Walter Spencer Anderson Griffith, M.D.
Alfred Theodore Rake, M.B., B.S.
Francis Charles Abbott, M.B., B.S.
James William Browne, M.B.
Edwin Harding Lendon, M.B.
George Redmayne Murray, M.D., M.A.
R. J. Bliss Howard, M.D.
Henry Jones Curtis, M.D., B.S.
Henry Albert Caley, M.D.
John Henry Dauber, M.B.
Theodore Henry Ionides, M.B., B.S.
Arthur Foxwell, M.D.
Horace George Turney, M.D.
Henry Grabham Lys, M.D.
Arthur Ernest Sansom, M.D.
John Henry Bryant, M.D.
G. F. Johnston, M.D.
Willmott H. Evans, M.D., B.S.
Charles Gibbs.
Robert Stephen Charsley.
Joseph Pearce Budgett Wills, M.D.
Arnold Lawson, M.D.

1894 Herbert Morley Fletcher, M.D.
Edward Wilberforce Goodall, M.D.
Harry Gilbert Barling.
Ernst Michels, M.D.
Charles Jerome Woollett.
StClair Thomson, M.D.
George A. Sutherland, M.D.
John Murray.
Richard Henry Anglin Whitelocke, M.B., C.M.
John Attlee, M.D., B.C.
Griffith Charles Wilkin.

1894 Thomas Hugh Dickson, M.B., B.C.
C. Gordon Brodie.
Bernard Scott.
John Porter Parkinson, M.D.
Thomas Rudolph Smith, M.B., B.C.
Arthur Pearson Luff, M.D.

1895 Herbert Murray Ramsay.
Arthur G. Phear, M.D., B.C.
Ernest E. Lewis, M.D.
Karl Grube, M.D.
Gerald R. Baldwin.
James Galloway, M.D.
F. B. Willmer Phillips, M.B.
Charles Henry Hough.
Leonard George Guthrie, M.D.
James Ernest Lane.
Frank J. Wethered, M.D.

1896 Richard Ackerley.
Charles Spurrell.
Cuthbert Sidney Wallace, M.B.
Charles R. Box, M.D.
Walter Stacy Colman, M.D.
Edward Ward, M.B.
Norman Dalton, M.D.
Fred. Parkes Weber, M.D.
Francis James Steward, M.B., B.S.
William Bezly Thorne, M.D.
Douglas Drew, M.D., B.S.
Surg.-Capt. W. R. Crooke-Lawless, A.M.S., M.D.
William Elliot Risdon, M.D.
Robert Milne Beaton, M.B.
Priestley Leech, M.D.
Ebenezer Stanley Smith, M.D.
G. H. A. Comyns Berkeley, M.B.
Edward Cooper Bentham.
Robert Herbert Mills-Roberts.
Jonathan Hutchinson, jun.
Frederick E. Batten, M.D.
Rutherford John Pye-Smith.
George Templeton.
Bertram Louis Abrahams, M.B., B.Sc.

1897 Raymond H. P. Crawfurd, M.D.
John Henry Deane.
Percy Furnivall.
Arthur Whitfield, M.D.
Frederick William Forbes Ross, M.D.
Harold Low, M.B.
Charles Ralph Keyser.
George Bertram Hunt, M.B.

1897 Hugh Roger-Smith, M.D.
Alfred Herbert Tubby, M.S.
Charles Lamplough.
H. Campbell Thomson, M.D.
Alfred P. Hillier, M.D., C.M.
F. Sherman Toogood, M.D.
E. Claude Taylor, M.D.
Charles Humphrys.
Charles Sempill de Segundo, M.B.
Arthur John Whiting, M.D.
Sir Alfred Downing Fripp, C.B., M.V.O., M.S.
Chichester Gould May, M.D.

1898 J. J. Perkins, M.D.
James Jackson Clarke, M.B.
Ernest Herbert Cobb.
Sydney Backhouse Hulke.
Thomas Herbert Kellock.
William Darley-Hartley.
Robert Jones.
Arthur Philip Beddard, M.B.
Alfred Ernest Russell, M.D., B.S.
William Cecil Bosanquet, M.D.
Jonathan F. C. H. Macready.
Arthur A. H. Partridge, M.B., M.Ch.
Leonard Avery.
Herbert Challice Crouch.
William Turner, M.B., B.S.
Harold Batty Shaw, M.D.
Joseph Stewart Richards, M.D., B.S.
William Henry Breffit Brook, M.D., B.S.

1899 George Edward Rennie, M.D.
V. Warren Low, M.D., B.S.
Rayner Derry Batten, M.D., B.S.
Charles E. Forbes Mouat-Biggs.
Rutherford Morison.
R. Murray Leslie, M.D.
Herbert Tilley, M.D.
Charles Owen Fowler, M.D.
Cyril Ogle, M.B.
Charles Herbert Fagge, M.S.
Claude Somerville Hawkes.
Henry Stephen Sandifer, M.D., B.S.
Marcus Marwood Bowlan, M.B.
Alfred Eïd, M.D.
Karl Fürth, M.D.

1899 Wm. Gream Stone, M.B.
Andrew Duncan, M.D.
Edmond Lucien De Chazal, M.D.
Henry Roe Walker.
Gustave Schorstein, M.B.
Edward Collingwood Andrews, M.D.
1900 Berkeley George Andrew Moynihan, M.S.
Harry Cooper, M.D.
George Augustus Tullis, M.D.
Griffith Lloyd Roberts, M.B., C.M.
Edward Percy Paton, M.S.
Kenneth Scott, M.D.
George Steele Perkins, M.D.
John Arthur Hayward, M.D.
Wilfred J. Hadley, M.D.
John Fawcett, M.D.
Arthur Bousfield, M.B.
J. S. Risien Russell, M.D.
Cecil H. Leaf, M.B.
Josef Paul Zum Busch, M.D.
1901 Ernest George Leopold Goffe, M.D.
Arthur Hamilton Nicholson Lewers, M.D.
Charles Reissmann, M.D.
Adrian Caddy, M.B.
Herbert William Carson.
Thomas Henry Morse.
Laurie Hugh McGavin.
F. U. Menzies.
Thomas Rupert Hampden Bucknall, M.S.
Edward Angas Johnson, M.B.
Percival Horton-Smith, M.D.
Sir Hugh Reeve Beevor, Bart., M.D.
Theo. N. Kelynack, M.D.
Walter S. Frith, M.B.
Thomas Carwardine, M.S.
Bowie Campbell Gowan.
1901 John J. Douglas, M.D.
Lieut.-Col. Alfred Keogh, C.B., M.D.
Harold Shuttleworth Barwell.
1902 George Beckett Batten, M.D.
Charles Henry Fennell, M.D.
H. L. Barnard.
Thomas Percy Legg, M.B.
Andrew Elliot, M.D.
Sir James Reid, Bart., G.C.V.O., K.C.B.
F. W. Price, M.B.
Alfred Ernest Sears.
Reginald F. Jowers.
Cecil Marriott, M.B.
Edward Farquhar Buzzard, M.B.
Donald John Armour, M.B.
Edred Moss Corner, B.Sc., M.B.
Herbert French, M.B.
S. Vere Pearson, M.B., B.C.
Lawrence Jones, M.B.
Thomas Crisp English.
Ernest Beddoe Hulbert, M.D.
Walter Fedde Fedden, M.B.
David Nabarro, M.D.
Charles A. Morton.
Victor Evelyn Collins, M.B.
Leonard S. Dudgeon.
R. O. Moon, M.D.
Charles Gordon Watson.
Charles Firmin Cuthbert.
1903 Raoul Ernest Delbruck, M.B.
Clive Riviere, M.D.
Edward A. Gates, M.D.
E. Graham Little, M.D.
Percy Robert Cooper, M.D., B.Sc.
Leonard Williams, M.D.
Ernest Lloyd Jones, M.D.
John Basil Hall, M.C.
Thomas Russell Atkinson, M.D.
Graham Simpson.
Edgar Beaumont.

REPORT

OF THE

COUNCIL OF THE CLINICAL SOCIETY,

MAY, 1903.

THE COUNCIL has to report the continued prosperity of the Society. The papers presented at its meetings have maintained that clinical character which it is the special object of the Society to promote, and many most interesting communications have been received.

The question of the disposal of the surplus volumes of the *Transactions* has been settled by the distribution of them to those members of the profession who applied, in consequence of notices in the medical journals to the effect that certain volumes might be obtained free of charge. Eighty such applications were received, and a total of 828 volumes were distributed. It was decided by the Council that after all applicants had been satisfied the stock of volumes kept for the Society should not exceed fifty copies of each year's work.

The number of Ordinary Members has now reached a total of 608. Twenty new members have been elected during the current year.

The ranks of the Honorary Members have been augmented by the election of some most distinguished physicians and surgeons, namely, Dr. Keen, of Philadelphia; Professor von Mikulicz, of Breslau; Dr. Osler, of Baltimore; Professor Erb, of Heidelberg; Professor Ernst von Leyden, of Berlin; and Mr. Timothy Holmes.

The Society has lost ten Ordinary Members through resignation and four from death, namely, Dr. W. H. Crosse, Dr. William Miller Ord, formerly a Treasurer of the Society; Dr. T. J. Maclagan, and Dr. J. W. Washbourn; and, in addition to these most prominent workers, the Society has to record the lamented deaths of Professor Rudolf Virchow and Dr. Carl Gerhardt, who were amongst the most honoured of its Honorary Members.

Mr. Charters Symonds has been appointed a Trustee in the place of the late Dr. Ord.

THE CLINICAL SOCIETY OF LONDON.

Statement of Cash Receipts and Payments from the 1st May, 1902, to the 30th April, 1903.

FREDERICK TAYLOR, Esq., M.D., *Treasurer.*

RECEIPTS.	£	s.	d.
Balance, 1st May, 1902	151	5	8
185 Subscriptions received by Collector £194 5 0			
200 ,, ,, Bank 210 0 0			
385	404	5	0
12 Subscriptions at 10s. 6d.	6	6	0
15 Admission Fees at £2 2s.	31	10	0
7 ,, ,, at £1 1s.	7	7	0
Sale of *Transactions*	15	6	8
Dividend on Consols (£640)	16	10	0
	£632	10	4

PAYMENTS.			£	s.	d.
Cost of *Transactions*, Vol. XXXV:					
Printing, paper, binding, delivery, &c.	£137	8 1			
Illustrations	67	15 0			
			205	3	1
Meetings:					
Rent of Rooms	105	0 0			
Hire of Microscopes and Lanterns	4	6 0			
A. Tapson (attendance)	7	0 0			
Refreshments	33	8 0			
			149	14	0
Treasurer and Hon. Secretaries:					
Secretarial assistance	26	5 0			
Commission to Collector	15	15 0			
			42	0	0
Stationery and Printing	84	13 8			
Diplomas to Hon. Members	2	12 6			
			87	6	2
Cost of Nominal Transfer of Consols (£640)			0	8	9
Cost of Power of Attorney			0	11	6
Return of Subscriptions paid in error			5	5	0
Petty Cash Expenditure			10	12	10
Balances:					
At Bank	128	16 7			
Petty Cash	2	12 5			
			131	9	0
			£632	10	4

HOWARD MARSH, *President.*
FREDERICK TAYLOR, *Treasurer.*
ANTHONY A. BOWLBY, *Hon. Secretary.*

Examined with the vouchers and found correct,
FREDERICK E. BATTEN,
STEPHEN PAGET, } *Auditors.*
May 1st, 1903.

PRESIDENTIAL ADDRESS

DELIVERED AT THE

CLINICAL SOCIETY OF LONDON

ON OCTOBER 10, 1902,

BY

HOWARD MARSH,

PRESIDENT.

GENTLEMEN,—I have to most heartily thank you for the great honour you have conferred upon me by electing me for a second year as your President.

Sir Thomas Watson, on taking the chair in January, 1868, at the first meeting of this Society, whose constitution he had helped to frame, and in whose success he showed so deep an interest, gave a presidential address; and his successors have, in almost every instance, followed his example. The addresses have been, I observe in looking over them, of two kinds. Several have been mainly devoted to some subject of medical or surgical interest; they have been, in other words, medical or surgical papers, which, in one or two instances, have been followed by a full discussion; while others have dealt chiefly with the functions of the Society, and have aimed at indicating how its objects can best be promoted. Undoubtedly communications from such men as Watson, Paget, Gull and Jenner, and others among your past presidents on any professional subject must always be alike valuable and welcome in the highest degree; but anyone who follows them may well hesitate to provoke invidious comparisons. At any rate, on considering the matter I have thought

that as those who took part in the establishment of this Society are becoming yearly more limited in number, and as many new members are taking their place, it is advisable that our original programme should be kept well in view. So that an address of what may be termed a business character, the purpose of which should be to consider in what directions advances are most likely to be made and by what kind of work they are most likely to be secured, may be appropriate to-night.

The Society is just entering on its thirty-sixth year. Are we on the right road and up to date, or does experience show that some modification of our constitution and of our methods is required, so that the future shall be as useful and satisfactory as the past undoubtedly has been? I do not, of course, mean the remote future—that we must leave. As to it, there is only one thing of which we may be certain,—that as the years roll on, so will also the ever-growing tide of scientific progress. What future advances will be, and whence or in what order they will come, no one, as yet, can tell; but in the immediate future, in the time with which we have to deal, our part is to keep an eye on the work that is next to be done and to study how it can be best carried out.

In retrospect we see that the lines on which the Society was founded were conspicuously wise and sound. The Society was established for a particular purpose. It was to cultivate what Sir James Paget in his address in 1870 termed clinical science,—that is, the scientific study of medicine and surgery from a clinical point of view, to be carried out by accurately recording and fully discussing the phenomena of individual cases and their treatment. It is in this specific and clearly defined programme that the strength and stability of the Society consist; for it fully accords with the stage of evolution which medicine and surgery have reached at the present time,—the stage, that is, of observation and analysis.

It is often said that medicine is not an exact science. Such an assertion is in itself unscientific. The word "exact" seems by a mental confusion to be used for some such term as complete or fully developed. Medicine, as the most complex of all the departments of biology, is obviously a subject which is pre-eminently suited for scientific investigation. Nor can it, with any show of justice, be maintained that the methods by which medicine is being studied are unscientific. On the contrary, founded as they are on anatomy, biology, chemistry and pathology, and involving

as they do the constant observation of vital phenomena of a highly complex order, they obviously fulfil all the conditions of scientific research. No doubt to the public the progress of medicine, tested by individual cases, must often seem inappreciable. They see that there are many forms of illness which can no more be cured to-day than they could twenty years ago. An estimate, however, based on such evidence is of course fallacious, since it ignores the numerous and fundamental advances which have taken place in other directions —advances which, in such a society as this, I need not particularise.

Nevertheless, we should ourselves be the first to own that progress is after all comparatively slow, and that considering the labour expended there is often but little to show. And progress is thus slow, not because our methods are defective, but because the whole subject involves a difficulty which, in other cases, is present in a much more limited degree,—the difficulty, namely, of ascertaining laws and formulæ which are capable of general application. The mathematician, the astronomer, and the chemist have been able to discover many general laws and formulæ which, true in one case, are true in every other also. They are, so to say, ultimate and independent facts which are liable to no disturbance or qualification. Thus the nebular theory, originally propounded by Kant, and confirmed and developed by Laplace and Herschel, is true not alone in the instances which these illustrious observers had in view, but in innumerable other examples in our solar and in other systems. At a single bound the mind in such a case escapes from the trammels of detail and stands face to face with an all-embracing and stupendous law.

In biology and its offshoot, medicine, it is different. Here the student is dealing with the phenomena of life itself. In biology—the science of the organism in its normal condition—these phenomena present themselves in endless variation; while in medicine not only is the normal replaced by the abnormal as the result of structural defect and methods of compensation, vicarious action, and so forth, but by the disturbance due to the introduction of external agents, which are numerous and variable, and whose action is modified by variations in the particular organism in which they have implanted themselves. The result, therefore, is, that while in the other sciences—for instance, astronomy and chemistry—the investigator has solid ground on which to stand and from which to advance, the student of medicine finds that the data upon which he has

to rely in his endeavours to solve the problems which he encounters are subject to such variation and disturbance that he is constantly thwarted, and finds any broad conclusion very difficult to reach, any general law extremely difficult to establish. Yet he is stimulated by the knowledge that, difficult as his task is, he is employing the same methods as those which, in other fields of research, have yielded earlier fruit, and which will assuredly in time be fully rewarded.

I have ventured on these crude remarks in order to indicate the stage which the study of medicine has at present reached, and to point out that happily our Society is working in close correspondence with it. Every case brought before us deals with carefully observed matters of fact, and as each paper is short there is the great advantage that the facts conveyed stand out singly and clearly defined, so that they are easily grasped and discussed, with the result that some definite conclusion can be reached. We are thus following Harvey in his revolt against the system of the old schoolmen who, instead of keeping to matters of fact, floated about in clouds of empty disputation; and we obey the injunction of Hunter—"Don't think" (he meant, of course, don't indulge in any mere speculation; don't put imagination in the place of knowledge and experience); "go and see."

The present stage of medicine, we may be sure, will be succeeded by a period, so to say, of synthesis or construction, in which it will be possible more often than now, and on the basis of knowledge which we are engaged in accumulating, to formulate general laws in regard to disease and, as to treatment, to replace empiricism by methods resting on exact knowledge of both the disease concerned and the agents to be employed in opposing it.

I must add a few words on the other side. Unless I do so I shall be thought to have rendered very scant justice to the great progress which medicine has beyond all question made in the last twenty years. Setting surgery aside, and confining attention entirely to medicine, it is familiar to all that wholly new lines of research have been opened up which have already yielded results of the highest value, and which hold out rich promise for the future. My remarks are intended to be general, and as such it will, I hope, be apparent that in many departments of medicine and surgery alike they are consonant with matters as they at present stand. My object in laying them before you has been to emphasise the manner in which this Society in the future, as has been

the case in the past, should do its work. We must keep strictly to the clinical side of medicine and surgery, and study individual cases; and we must also, in most instances, keep to our time-limit of about ten minutes. Papers which are much longer, and papers on general subjects, valuable as they are, are not appropriate here; and to accept them would be to interfere with the functions of other societies, and to take what does not properly belong to us.

On thinking over the ordinary working of the Society, one is struck by the fact that the subjects brought forward for discussion are determined to a large extent by accident. Papers are suggested by the cases which happen to come under the observation of individual members. And from this it results that the Society takes little part in the discussion and settlement of the important questions which from time to time challenge the general attention of the profession. That the study of individual cases should be our chief business is clear. But it may be worth while to consider how far it is advisable to exercise any kind of selection of subjects for discussion.

Would it be useful to appoint small departmental committees whose duties it would be—

1. To suggest to the Council certain subjects, that were at the time attracting general attention, for discussion;

2. After the discussion, to consider what points had seemed most important and most in need of further study, and to take these points into consideration and report later upon them?

For this treatment, *e. g.*, (on the medical side) acute rheumatism and (on the surgical) intra-peritoneal suppuration might be selected. Such subjects as these are fair examples of many others in regard to which divergent or even contradictory opinions as to treatment are held; and methods of treatment become current and, by repetition and strong colouring by friends and opponents, grow more and more involved and confused, so that lookers-on are left in a condition of embarrassing uncertainty. In these circumstances a discussion at this Society, if carefully arranged and regulated so that those most competent to express opinions were invited to assist, might be attended with valuable results. General discussions in former sessions have, as all will remember, taken place—on Charcot's disease, and on myxœdema and other subjects,—which were undoubtedly very useful. But such have been—like the papers on separate cases—deter-

mined very much by accident. By the plan I suggest such discussions would be on carefully selected subjects, and, as they would take place probably not more often than once in every session, they would not materially interrupt our ordinary work.

The subsequent reports of the sub-committees concerned would be useful by emphasising, as I have said, the points of most importance and indicating the stage of evolution down to which they had been brought. Needless to say, definite conclusions, to be sound, can be arrived at only when the necessary materials of forming them have been acquired. But often uncertainty does not depend so much on want of knowledge as on the fact that the subject has not been considered in all its bearings by those who approach it with an open mind and, so to say, in the capacity of a special jury.

Our clinical evenings, I feel sure all will agree, are a great success, and the interest taken in them is shown by the large attendance of members.

COMMUNICATIONS.

I.—*The Nasal Treatment of Asthma.* By Alexander Francis, M.B., B.C., B.A. (Cantab.), M.R.C.S.Eng., Brisbane, Queensland. *Read October* 10, 1902.

SINCE the days of Aurelian the association of asthma and nasal disease has been noticed by various writers, but it was not until Voltolini in 1871 published 11 cases of the relief of asthma by removing nasal polypi, that any great attention was paid to the connection. Professor Hack, in 1884, brought matters to a more practical issue by endeavouring to establish an intimate relationship between asthma and nasal lesions. Following in their footsteps many observers were able to record successful results in the treatment of asthma by nasal means, and the belief in the nasal origin of asthma gradually gained ground until a climax was reached when Bosworth, of New York, stated in 1886 that "a large majority of, if not all, cases of asthma are dependent upon some obstructive lesion in the nasal cavity." In 1889 he published the list of 46 cases of asthma, of which 28 were cured and 12 improved by intra-nasal treatment. Of these, 17 had suffered from polypi, and the rest from hypertrophic rhinitis, deflected septum, or rhinitis with adenoid vegetations. Later he reported 88 cases of asthma, of which 42 were cured and 33 improved. About the same proportion of cures was obtained by Schmiegelow and Heyman. In Schmiegelow's 50 cases of asthma 32 were cured and 11 improved; but among the 32 "cures" asthma recurred in 17 cases, with a recurrence of the nasal lesion. In the 53 cases treated by Heyman

29 were cured and 14 improved. Lubinski, on the other hand, only claimed to have cured 27 and improved 13 out of 143 cases of asthma with nasal and pharyngeal lesions. Bosworth's belief that the majority of cases of asthma have some nasal lesion has been strongly combated, and the general opinion now seems to be that, even in such cases where nasal trouble is found, little hope of obtaining permanent relief can be expected from nasal treatment. The most recent text-books on medicine, in dealing with the treatment of asthma, merely mention that the naso-pharynx needs attention if any gross lesion be found; but all hope of obtaining relief is placed on treatment by climate, diet, and drugs. It has been further pointed out that the rhinologist is apt to be misled in his estimate of the frequency of nasal lesions in asthmatic subjects, as he is only likely to meet with such cases as have some subjective nasal symptoms. In the 'Report of the Proceedings of the Laryngological Society of London,' May 5, 1899, on a discussion on "Asthma in its Relation to the Upper Air-passages," which was opened by Dr. Percy Kidd and Dr. P. McBride, and in which many of the leading rhinologists took part, it can be seen that the general opinion as regards the prospect of obtaining permanent relief was anything but hopeful. Sir Felix Semon summed up the results of his experience of the nasal treatment of asthma by dividing the cases into three classes: (1) lasting success obtained—exceedingly small percentage; (2) temporary benefit—comparatively large percentage; (3) no success at all—very large percentage. To which Dr. StClair Thomson suggested the addition of a fourth class, viz. those who were considerably damaged by intra-nasal treatment. Although some speakers took a much more hopeful view and were able to record numerous cases of successful results, still the consensus of opinion was fairly expressed by Dr. Percy Kidd when he stated that "if there was obvious disease of the nose local treatment was advisable, though the uncertainty of the result as regards the asthma should be clearly explained to the patient." This brings me to the point that I wish especially to deal with. No speaker suggested the advisability of treating the nose in cases of asthma where there was no obvious nasal lesion; and yet such, in my opinion, are the very cases where the most hopeful prognosis can be given. The observation that the rhinologist is likely to see only such asthmatics as are conscious of some nasal trouble, does not apply to me, because

the great majority of the cases I have seen, consulted me because I was known to treat asthma, and not because I practised rhinology. My experience, from treating over 400 cases of various kinds of asthma, is responsible for the opinion I have expressed that it is those asthmatics which present no gross nasal lesions, and where there are no subjective nasal symptoms, that give the best hope of affording relief by intra-nasal treatment. I quite agree with the remark that one often hears, that where there are polypi or other gross nasal lesions, there is a prospect of affording relief by surgical treatment, although it is necessary to explain to the patient that the relief may not be permanent, and that there is a probability that there will be no relief at all; but I maintain, and the more experience I get the more strongly do I maintain, that these are the only cases of asthma where such a poor prognosis need be given. I am fully conscious that such a statement is almost sufficient to condemn me to be no further heard, but I would submit that the record of cases that I beg to lay before you justifies my belief. Of the 402 cases here recorded, 14 only, that I know of, obtained no relief; and of these, 3 suffered from polypi and 2 from such pronounced deflections of the septum that it was impossible to apply the galvano-cautery without doing operations, which were declined. Only 8 cases that presented no gross nasal lesion, out of 346 of such treated, obtained no relief, and of these 3 were only seen by me three times, and 1 four times. Of the remaining 4 cases, 1 was a confirmed morphomaniac, which may have influenced the result, and it is curious to note that in 2 of the remaining 3 cases I could at any time produce an asthmatic spasm by touching certain parts of the nasal mucous membrane, pointing to an abnormally sensitive reflex mechanism. In addition to these 14 known failures there are 17 cases where I have been able to obtain no record of the result, and a number of others that were completely relieved, or greatly improved, were lost sight of. This is particularly unsatisfactory, as many cases that I have at times looked upon as failures, in consequence of having lost sight of them after a few treatments, months or years later I discovered had not come back to me because they had obtained complete relief. From the experience I have obtained in treating these cases I have come to the following conclusions:

1. That asthma is due to reflex spasm of the bronchial tubes.

2. That the irritation may originate in the nose.

3. That asthma is not directly due to any mechanical obstruction of the nasal passages, and is not commonly caused by any gross nasal lesion.

4. That some part of the nasal apparatus has a controlling influence upon the respiratory centre; or there is in the nose, as it were, an agency through which the afferent impulses must pass.

1. I feel that it would be an impertinence on my part to offer any suggestions on the pathology of asthma. The commonly accepted theory is that the dyspnœa of bronchial asthma is due to spasm of the bronchial muscles. Nearly every case that I have seen has tended not only to maintain this theory, but also to confirm my belief that the spasm is produced by reflex irritation. Also I would not make the exception of cardiac and renal asthma, which are usually distinguished from bronchial by having a pathology of their own. But I shall have occasion to refer to this later.

2. That the irritation may originate in the nose is to be inferred from (1) the intimate association between hay fever and asthma; (2) the very common record of excessive sneezing at some period in the previous history of an asthmatic patient; (3) the not infrequent alternation between asthma and sneezing.

Such cases as the following demonstrate the fact more definitely:

No. 391, a boy æt. 9, was perfectly well until he fell on an oil drum and broke the bridge of his nose. Asthma developed immediately afterwards, and continued in a severe form for five and a half years. Under nasal treatment he obtained instant and marked improvement.

In Case No. 97 the first sign of asthma dated from a horse kick which broke the patient's nose. When I first saw him the asthma was very severe, but was completely relieved by cauterising his septum nasi.

In Case No. 309 (W. E.) severe sneezing followed an operation on his nose. This was succeeded by attacks of bronchitis and asthma on the slightest provocation. In this case the wheezing was speedily relieved by treatment, but the attacks of sneezing persisted for some time.

In Case No. 4 (E. W.) an operation on the nose was also responsible for the onset of asthma. A country practitioner had occasion, nine years previously, to perform what he described as "amputation of the middle turbinated." After-

wards, to stop the hæmorrhage, he found it necessary to plug the nostril so tightly each day for a fortnight that extreme deflection of the septum and almost continuous asthma resulted. When I saw the patient, temporary straightening of the septum at once relieved the bronchial spasm, but he declined any further surgical interference.

Other cases pointing to the nasal origin of asthmatic spasm are Nos. 143 and 177, in both of which touching the septal mucous membrane instantly induced bronchial spasm, which was as quickly relieved by painting the part with a solution of cocaine. No. 143 (T. R. S.) thus described the sensation of my touching his septum :—"It felt as if you were screwing my chest up in a vice." Again, I have frequently met with cases where the patient could prognosticate an asthmatic attack by the onset of an irritation in the nose. Nos. 47 and 141 are examples.

Lastly, I have not infrequently brought on a severe attack of asthma by cauterising too severely or too frequently, as one is sometimes tempted to do when patients come from a great distance and have only a short time to spare.

3. It is true that nasal polypi and other obstructive lesions of the nose are found in conjunction with asthma, but I believe the association is not so common as is generally supposed. Schmiegelow found that in 139 cases of nasal polypi only 31 had asthma, and in 517 cases of chronic rhinitis 40 had asthma. In my 402 cases of asthma 32 only had polypi, and 24 had other marked obstructive lesions. I believe that when asthma and polypi occur together they are more probably the result of some common factor than that they have any causal relationship. Of the 32 cases of asthma with nasal polypi that I have treated, one only obtained complete and permanent relief, and in this case, No. 52, I did not touch the polypus. He was one of the worst asthmatics I have seen, and had suffered acutely for thirty years. On his first visit to me, whither he was brought rather against his will, as he had long lost all faith in treatment, I described him in my notes as "a perfect wreck." Finding a polypus in his nose, I felt no hope of affording him any material relief, and fearing lest I might possibly render his condition worse by removing the polypus, I contented myself with merely cauterising his septum. He returned to his home in Victoria a few days later, and three years afterwards wrote to tell me that, with the exception of a slight attack after a very long journey in Western Australia, he had remained perfectly

free from asthma. I saw him last April, when he still had the polypus, but no sign whatever of asthma. In this case evidently the polypus was not the cause of the asthma. Again, in each of the three cases where no relief of asthma was obtained from treatment, there was no recurrence of polypi after their removal, and in two of these cases (Nos. 25 and 154), in spite of the nasal passage having been rendered free, which previously had been completely obstructed, the asthmatic spasm became intensified after the polypi were removed. I have frequently been struck by the difficulty an asthmatic patient experiences in breathing through the nose, even when there is no mechanical obstruction.

Case No. 203 suffered from atrophic rhinitis, and yet complained of inability to breathe through the nose when suffering from an attack of asthma.

No. 384 (Mr. B.) is a more striking example. When I first saw this patient he was suffering from severe dyspnœa, and was quite unable to breathe through his nose. On examining him I found both nostrils completely obstructed by engorged turbinated bodies. On applying extract of suprarenal capsule to the turbinateds I rendered the passages so free that the pharyngeal vault could be easily seen through them. Still the patient felt no improvement, and characteristically sucked in his *alæ nasi* in his forced vain attempt to draw air through his nose. I then painted part of his septum with a drop of solution of cocaine on a pledget of cotton wool, which did not mechanically enlarge the passage to any appreciable extent; but instantly the patient experienced the greatest relief, and stated that he could now breathe quite freely through his nose. For curiosity I cauterised his septum before treating his turbinateds, with the result that his dyspnœa was relieved before his nasal passages were rendered free.

C. A. Parker's experiments, showing that inspired air travels through the middle rather than the inferior meatus, are particularly interesting in connection with such cases, which also indicate that the nasal meatus are more than mere aëroducts.

4. That the nose should have the power of influencing afferent impulses to the respiratory centre is to me the most interesting phase of the whole phenomena of asthma. If it be so, then it matters not whether the originating irritation be bronchial, gastric, cardiac, or renal, the resulting spasm can be profoundly affected by intra-nasal treatment. This

belief has been forced upon me by the behaviour of many cases, of which the following are examples:

No. 23 (Mr. E. A. S.) suffered constantly from asthma, although he consumed large quantities of potassium iodide. He stated that for five years he had not slept for more than two hours at a time without having recourse to smoke inhalations, and if he touched a drop of beer or ate certain foods he would be fixed to a chair so closely that if his house took fire he could not move out of it. He was indignant when I suggested looking at his nose, because he was convinced that all his trouble arose from his stomach. His nasal organ appeared quite normal; nevertheless he slept uninterruptedly for ten hours the first night after I cauterised his septum. Then, as so many asthmatics do, he sought to test the efficacy of the cure by drinking beer and eating such food as he had found in his previous experience certain to bring on an attack; but up to the present (four years) he has failed in his attempts.

No. 87 (Mr. H.) was likewise convinced that his asthma was due to indigestion following malarial fever. He had been a terrible sufferer for nine months, during which time he had lost four stone in weight. His asthma was always worse after taking food, and particularly severe after eating certain things. He had visited each of the Australian states without obtaining relief, and when I first saw him was not able to walk many yards without resting. He derived no benefit from the first two occasions on which I cauterised his septum, but after the third burning the relief was absolute. The same day he ran to catch a train, and at the end "was not," to use his own words, "puffing enough to blow out a match." On his next visit he told me he had left all his asthma in my room, and said it "is not only miraculous, but ludicrous."

Such cases are so common that I have come to look upon the remark "all my trouble comes from my digestion" as one of the most hopeful symptoms in forming a prognosis. More striking still are the cardiac cases.

No. 17 (Mr. S.) suffered from old-standing heart disease, and when I saw him was confined to his bed, but unable to lie down on account of severe dyspnœa. His wife told me that the noise of his wheezing at night disturbed the whole household. After cauterising his septum nasi his orthopnœa left him, and did not return during the three months that he continued to live.

No. 71. Mr. E.'s asthma appeared to be of purely cardiac origin. Although he had no cardiac murmur, the heart-sounds were weak and irregular. He could make no exertion and not even walk many yards without bringing on distressing dyspnœa, which, however, was soon relieved upon his resting. After a few treatments he was able to walk uphill with ease, and the last time I saw him he walked three miles and showed no sign of breathlessness. He told me he spent most of his time now digging in his garden.

Among other apparently cardiac cases are Nos. 26, 96, 112, and 159.

Many cases could be quoted where asthma seemed to originate from irritation in the bronchial tubes or lungs themselves, but I shall merely mention one or two where the chest trouble was so pronounced as to have given the impression that the patient suffered from phthisis.

No. 6 had very severe asthma for over two years, and needed to have Himrod powder burning in the pulpit to allow him to preach. He had lost 18 lbs. in weight in ten months, and, as far as the asthmatic disturbance in his chest would allow one to tell, had the physical signs of phthisis, for which he had been treated. His cough was particularly distressing, and he expectorated a large quantity of phlegm. Fifteen months previously, before I had discovered the far-reaching effects of cauterising the septum, I had removed a large septal spur from his right side without giving any relief to his dyspnœa. On cauterising his septum, not only every sign of asthma, but all chest trouble disappeared. I saw him again last April, five years later, when he told me he had been in the most robust health ever since, and had gained two stone in weight.

No. 45 (Miss H. E.) had suffered from asthma with signs of tubercle, including cough, hæmoptysis, and loss of weight, for four months. Under nasal treatment she obtained complete relief of asthma and all chest trouble, and more than regained her lost weight.

Also, there are many cases where tubercle undoubtedly did exist in conjunction with asthma, in which the dyspnœa was completely relieved by cauterising the septum.

The only case that I have seen where asthma seemed to depend directly on renal disease was No. 180 (Mr. O.). He suffered from chronic Bright's disease, and for nine years had periodically experienced severe asthmatic attacks, which were coincident with exacerbation of his renal trouble. Under

nasal treatment his asthma was very greatly relieved, and although he had other attacks later, the interval between them was much longer and they were never severe.

I should like to draw attention to some other cases which further exhibit the influence of certain parts of the nose upon the mechanism of the bronchial innervation.

No. 238 (Mr. M. M.) was sent to me from the north of Queensland with a note in which the writer doubted whether the patient would reach Brisbane alive. He was in the direst distress when brought to me, and on listening to his chest it seemed wonderful that he could keep alive on the small quantity of air that appeared to enter his lungs. His nose was apparently normal, but although the passages were very free his painful gasps for breath were all taken through the mouth. On cauterising the right side of his septum nasi he experienced instant relief in the same side of his chest, and exclaimed, "You have loosened something here," pointing to the region of his liver.

Patients have frequently complained that their tightness in breathing was only on one side of their chest, and also frequently have I heard them state that my cauterising one side of their septum gave relief on the same side of their chest. A common expression of patients is, "You have untied something at the bottom of my ribs," and almost invariably is the sensation of the cauterising referred by them to the free border of the ribs, and not to the nose. Several times it has only been by showing a patient the length of my cautery point that I could convince him that it was impossible for me to have actually touched the spot where he felt the sensation. Still, I never had such ocular demonstration of the unilateral effect of the cautery as in this case. The right side of the chest could be seen to rise and fall, while the left appeared bound down almost motionless. So extraordinary was the effect that I had his chest stripped again to listen. Whilst no air appeared to be entering the left lung, on the right side it was passing in freely amidst a great chorus of crackling râles. Although the man begged me to "undo" the left side of his chest at once, I thought it better not to touch him again until the following day. When he came on the next morning he was still breathing in the same extraordinary manner. He stated that he had slept well, that the right side of his chest had continued perfectly free, but the left had been very tight and painful. This latter was similarly released by the use of the cautery, and the man remarked he could

now breathe more freely than he had done for years. He remained under observation for some weeks, and in September, 1901, I received a report that he had remained well and was working on a sugar plantation.

No. 74 (Miss M.) represents another class of case. She had suffered for two years from distressing dyspnœa, especially on any exertion. Her breathlessness had been attributed to a large unilateral goitre, and she had been told that nothing but its removal could give her any relief. However, on cauterising her septum nasi the dyspnœa was completely removed, and she had ever since worked as a cook without any discomfort.

No. 345 (Mr. T. McG.) was sent to me from the country on account of a supposed growth in his throat causing dyspnœa. The latter became so severe after coughing that his friends had at times thought him dying. Finding upon examination that there was nothing really abnormal in his throat I cauterised his septum nasi, with the result that his "lump" instantly disappeared, and he breathed quite freely. Curiously, he developed later a wheeze, which he referred to the left side of his chest, but this, too, yielded to further treatment.

Thus I have attempted to show that there is an area on the septum nasi which is intimately connected with asthma, and by treating which one is able, in the great majority of cases, to relieve all bronchial spasm. I am fully conscious that the galvano-cautery is a crude and elementary means of treating the part, and I quite believe that before long other more efficacious methods will be found. My hope is that these notes, by drawing attention to this part of the nose and its connection with asthma, will do a little towards evolving a remedy that will invariably give relief to one of the most distressing of all complaints. In the meantime, while such results as I have here recorded can be obtained by the imperfect but simple and painless galvano-cautery, we need not take the desperate view that has been expressed by an eminent authority, that the asthmatic's only hope in this world lay in potassium iodide.

Summary.

Total number	402 cases.
Nose apparently normal . . .	346 „
Polypus	32 „
Other gross lesions	24 „

Complete relief obtained . . .	194 cases.
Complete relief till lost sight of, or still under treatment . . .	30 „
Great improvement	73 „
Great improvement till lost sight of, or still under treatment . .	50 „
Temporary relief	20 „
Slight relief	4 „
No record	17 „
No relief	14 „

Males 282; Females 120.

APPENDIX.

N.B.—The severity has been denoted by numerals as follows:

1 = Asthma only *at times.*
2 = Asthma *every night, or frequently each week.*
3 = Asthma *constant, day and night.*
3 + has been used occasionally *when the asthma is especially severe.*

1. Mr. E. R., æt. 24, Nov. 24, '96. Variety, spasmodic; duration, 10 years; severity, 3. [One cauterisation; complete relief.*] Breathing never quite free; dyspnœa on any exertion; severe attacks frequently. *N.B.*—Septum cauterised with view to subsequent operation on ridge, which, however, was found unnecessary, as all signs of asthma ceased. Septal ridge on left.

2. Mr. F. S. R., æt. 30, Dec. 14, '96. Variety, spasmodic; duration, 30 years; severity, 1. [Four sittings; complete relief.*] Brother of Case No. 1. Bad attacks of asthma frequently; quite free in intervals. Septal ridge on left.

3. Mr. A. K. C., æt. 46, Feb. 22, '97. Variety, spasmodic; duration, 10 years; severity, 1. [Four sittings; complete relief.*] Attacks frequently depending chiefly on locality and sudden changes of atmospheric condition. *N.B. After treatment.* Dec., '98.—No trace of asthma since Feb., '97. Oct., 1901.—Fresh attack of asthma after influenza, relieved by one treatment.

4. Mr. G. W., æt. 23, Feb. 23, '97. Variety, spasmodic; duration, 9 years; severity, 3. [Four sittings; no relief.*] Asthma constant; began after operation in the country for removal of left mid. turbs.; the prolonged after-plugging needed caused extreme deflection of septum to right. *N.B.*—Temporary straightening of

* *The number of sittings and the result of treatment should be read at the end of each case.*

septum relieved asthma; patient declined operation. Deflection of septum to right.

5. Mr. H. S., æt. 19, Mar. 2, '97. Variety, spasmodic; duration, asthma 18 months, cough 15 years; severity, 2. [Ten sittings; complete relief.*] Asthma nearly every night, and frequently by day. *N.B. Under treatment.*—Relief at first, but asthma returned in May; then obtained complete relief. Dec. 28, '97.—No asthma since, with tubercle progressing. Septal ridge on left.

6. Rev. W. L., æt. 45, Mar. 12, '97. Variety, bronchial tubercle (?); duration, 2 years 4 months; severity, 3. Came on with severe bronchitis; much cough and phlegm; suspected tubercle; lost 18 lbs. in 10 months; needed Himrod burning in pulpit to allow him to preach; severe asthma almost every night. *N.B.*—Since Feb., '96, I removed septal ridge left, which enabled him to breathe more easily through nose, but did not affect asthma. On Mar. 12 he obtained instant relief on cauterising left septum. Feb., 1902.—Has gained 2 stone in weight; had no sign of asthma or chest trouble since. Septal ridge on left removed previously.

7. Mr. A. A., æt. 16, Mar. 18, '97. Variety, bronchial; duration, 1 year; severity, 1, 2. [Three sittings; complete relief.*] *N.B.*—Supposed to have tubercle.

8. Mrs. J. C. W., Mar. 20, '97. Variety, spasmodic; duration, 2 years; severity, 2. [Seven sittings; temporary relief.*] Severe attacks frequently; complete freedom from asthma only when pregnant. Many polypi.

9. Mrs. W., æt. 34, Mar. 24, '97. Variety, bronchial; duration, 8 years; severity, 1, 2. [Eight sittings; complete relief.*] Began with bronchitis; always much cough and phlegm; supposed phthisis.

10. Mr. A. B. B., æt. 45, April 7, '97. Variety, spasmodic; duration, 5 years; severity, 2, 3. [Five sittings; complete relief.*] *N.B.*—Galvano-cautery to right side increased asthmatic spasm; great relief after second treatment.

11. Mrs. M., æt. 30, April 21, '97. Variety, bronchial; duration, 8 months; severity, 1. [Four sittings; complete relief.*] Began after bronchitis; catches cold frequently, then has asthma.

12. Mr. D. McT., æt. 42, June 8, '97. Variety, bronchial; duration, years; severity, 1, 2. [Great improvement.*] Very severe attacks lasting 2 or 3 weeks, periodically. *N.B.*—Removal of large ridge on right improved freedom of breathing, but did not affect asthmatic attacks. Septal ridge on right.

13. Miss G., æt. 21, July 2, '97. Variety, spasmodic; duration, 1 year; severity, 1. [Twenty sittings; temporary relief.*] Came on after a cold; removal of polypi always affords temporary relief; polypi, situated far back, very small and quickly recurrent. Polypi both sides.

14. Miss F., æt. 16, July 7, '97. Variety, spasmodic; duration, 9 years; severity, 2. [Nine sittings; no relief.*] Hypertrophic rhinitis.

15. Mr. P. O. M., æt. 37, Nov. 6, '97. Variety, bronchial; duration, 3 years; severity, 3. [Seven sittings; temporary relief.*] Asthma constant during last 9 months; has had phthisis for some years. *N.B. Under treatment.*—Complete relief for a time after each removal of polypi. Polypi both sides.

16. Mr. C. D. P., æt. 29, Nov. 28, '97. Variety, spasmodic; duration, 12 years; severity, 2. [Five sittings; complete relief.*] *N.B.*—Removal of large septal ridge on each side, 15 months previously, gave no relief of asthma. Septal ridge each side.

17. Mr. W. S., æt. 70, Nov. 30, '97. Variety, cardiac; duration, 2 months; severity, 3. [One sitting, with complete relief of wheeze during rest of life (some months); could lie down.*] Old-standing heart disease; severe orthopnœa; cannot lie down or sleep; cardiac treatment gives no relief of dyspnœa.

18. Mrs. K., Jan. 3, '98. Variety, spasmodic; duration, 1 year; severity, 1. [Five sittings; great improvement.*] For 12 months before asthma began suffered from extreme hyperæsthesia and irritability of naso-pharyngeal mucous membrane.

19. Mr. T. W., æt. 40, Jan. 15, '98. Variety, bronchial; duration, 2 months; severity, 3. [Fifteen sittings; temporary relief.*] Began after prolonged wetting. Many polypi.

20. Mrs. S., æt. 34, Jan. 15, '98. Variety, spasmodic; duration, 3 years; severity, 2, 3. [Ten sittings; complete relief.*] Began after severe cold on chest. *N.B.*—After third treatment was quite well for 12 months; asthma returned after birth of a child. Complete relief again after 3 treatments till Jan., 1901; then asthma returned after birth of another child.

21. Mrs. K., æt. 40, Jan. 25, '98. Variety, bronchial tubercle; severity, 2. [Six sittings; complete relief of asthma.*] Asthma nearly every night; frequently very severe attacks; always short of breath. Came to Queensland 20 years previously for phthisis. *N.B. Under treatment,* Jan., '99.—No wheezing since; can walk better than she has done for years.

22. Mrs. W., æt. 31, Feb. 22, '98. Variety, spasmodic; duration, years; severity, 1, 2. [Six sittings; great improvement.*]

23. Mr. E. A. S., æt. 48, March 7, '98. Variety, ? gastric bronchial; duration, 5 years; severity, 3. [Two sittings; complete relief.*] Came on after bronchitis; has not slept 2 hours consecutively without using Himrod for 5 years; a glass of beer or any indigestible food always brought on a severe attack; much cough and phlegm.

24. Mr. J. O'C., æt. 30, April 21, '98. Variety, bronchial; duration, 2 years; severity, 2. [Five sittings; complete relief.*] Came on after bronchitis. *N.B.*—After 3 treatments complete relief till April, '99, when slight return; 2 treatments again afforded complete relief; has had bronchitis since, no asthma.

25. Mr. W. H., æt. 54, May 2, '98. Variety, spasmodic; duration, 20 years; severity, 3. [Six sittings; no relief.*] "Not a

night free for 20 years;" had polypi removed 15 years ago. Very large polypi, which I removed.

26. Mrs. S., May 4, '98. Variety, spasmodic cardiac (?); duration, 12 months; severity, 2. [Five sittings; great relief.*] *N.B.*—Died of cardiac failure 3 years later.

27. Mrs. W. C., æt. 37, May 7, '98. Variety, spasmodic; duration, 13 years; variety, 3 years. [Eight sittings; great relief.*] Had asthma intermittently for 5 years; almost constant during last 8 years, when Himrod necessary every night. Large polypus right.

28. Mr. S. T., æt. 20, May 7, '98. Variety, spasmodic; duration, 2 years; severity, 1. [Six sittings; complete relief.*] Asthma 3 or 4 times a month; previous history of hay fever. *N.B. After treatment.*—Asthma relieved, but sneezing returned, which, however, yielded to further treatment.

29. Mr. D. B., May 14, '98. Variety, spasmodic; duration, 7 years; severity, 1, 2. [Five sittings; great improvement.*] Has asthma at least once a week. *N.B. Under treatment.* June, '98.—No asthma since. July, '98.—Has had 1 attack. Dec., '98.—No attack since.

30. Mrs. A., æt. 30, May 14, '98. Variety, spasmodic; duration, all her life; severity, 3. [Three sittings; complete relief.*] Has had asthma since infancy; very bad during last 10 years; diet has direct effect on attacks. *N.B. Under treatment.* May 28, '98.—No attacks since, but twice had catch in breath, which coughing cleared.

31. Mr. R. M., æt. 26, May 17, '98. Variety, spasmodic; duration, all his life; severity, 2. [Six sittings; complete relief.*] Bad at æt. 14; then free for 3 years; since then wheeze constant; severe attacks at times; can do no active work. *N.B. Under treatment.* June 21, '98.—Can play tennis and bicycle without difficulty. Feb., '99.—Quite well all winter. Jan., 1901.—No sign of asthma.

32. Rev. J. C. H., æt. 75, May 17, '98. Variety, bronchial cardiac; duration, some years; severity, 2. [Ten sittings; improvement.*] Never quite free; occasional severe attacks, lasting several days. *N.B. Under treatment.*—Always gets relief for some weeks after each treatment.

33. Mr. W. F. K., æt. 40, May 20, '98. Variety, spasmodic; duration, 4 years; severity, 3. [Sixteen sittings; great relief.*] Asthma continued; has to smoke "asthma leaf" every night, often three times a night; has been unable to work for nearly 2 years; sneezes much; meat or beer invariably induces an attack. *N.B. Under treatment.* Feb., 1902.—Has been 3 months without an attack; can do light work.

34. Mr. H. M. P., æt. 32, May 24, '98. Variety, bronchial; duration, 6 months; severity, 3. [Four sittings; great improvement.*] Began with bronchitis; asthma gradually getting worse; sleeps in chair before fire; dyspnœa on any exertion; constant cough and phlegm. *N.B. Under treatment.* June 5, '98.—No asthma since; quite strong; can lift 200-lb. bag. Oct. 22.—Has had

influenza cough, and asthma returned during last 10 days; not seen since.

35. Mr. D. J., æt. 38, May 24th, '98. Variety, spasmodic; duration, 7 years; severity, 3+. [Eleven sittings; complete relief.*] Began with bronchitis; asthma constant since; unable to work; uses Himrod frequently by day and every night. *N.B.* Nov., '99.—Has worked in coal-mines all winter.

36. Mr. J. W., æt. 33, May 24, '98. Variety, spasmodic; duration, 3 years; severity, 2. [Five sittings; great relief.*] Wakes at midnight with choking; attacks last till morning. *N.B. After treatment.* June, '98.—One attack only since. Aug. '98.—Only 1 attack during last month.

37. Mr. A. A., æt. 45, May 31, '98. Variety, spasmodic; duration, 2 years; severity, 3. [Two sittings; relieved; lost sight of.*] Asthma been gradually getting worse; been confined to bed all winter. *N.B. Under treatment.* June 14, '98.—Great improvement; able to get about again.

38. Mr. W. C. G., æt. 47, June 11, '98. Variety, spasmodic; duration, 4½ years; severity, 3+. [Ten sittings; complete relief.*] Asthma came on after severe wetting in a flood; has not had a free breath day or night for 4½ years. "I will let you cut my throat if you will give me five minutes' peace in breathing first." *N.B After treatment.*—Great relief from first treatment; gained 1 stone in weight during the first month. *N.B.*—For the last 3½ years has been working as a blacksmith's striker.

39. Mr. R. A. C., æt. 50, June 13, '98. Variety, spasmodic; duration, 6 years; severity, 2. [Five sittings; great relief.*] Always bad with change of weather; attacks nearly every night; uses Himrod constantly.

40. Mr. J. C., æt. 33, June 16, '98. Variety, spasmodic; duration, 4 years; severity, 2. [Five sittings; complete relief.*] Four years ago very bad for 12 months; lost 2 stone in weight; better till a month ago, since when been very bad every night. *N.B. After treatment.* June, 1900.—Has had bronchitis since, but no return of asthma.

41. Mrs. H. C. P., æt. 35, June 22, '98. Variety, bronchial; duration, 4 years; severity, 3+. [Nine sittings; complete relief.*] Began with bronchitis; cannot lie down or take any exertion; severe wheeze constant; much cough and phlegm. *N.B. After treatment.*—Previously flatulence invariably brought on an attack, has had no effect. Septal ridge both sides (not operated on).

42. Mrs. A. C., æt. 52, June 22, '98. Variety, spasmodic, with tubercle; duration, 4 years; severity, 2. [Nine sittings; complete relief of asthma.*] Began after illness, with much coughing and hæmoptysis. *N.B. Under treatment.* Feb. 28, '99.—No asthma. March 10, '99.—Cough and much phlegm, with blood, but no asthma. June, '99.—Much cough, but no asthma. Jan., 1902.—Cough less; no asthma since.

43. Mr. J. H. R., æt. 38, June 24, '98. Variety, spasmodic; duration, 2 years; severity, 1, 2. [Five sittings; complete relief.*] Suffered previously from irritation of throat.

44. Mrs. G. F., æt. 32, July 28, '98. Variety, spasmodic; duration, 16 years; severity, 1, 2. [Eleven sittings; improvement.*] Suffers from paroxysmal sneezing, and frequent severe attacks of asthma. *N.B. Under treatment.*—Gradual improvement from the first. Nasal mucous membrane extraordinarily pallid.

45. Miss H. G., æt. 19, July 29, '98. Variety, spasmodic and tubercle (?); duration, 4 months; severity, 2. [Six sittings; complete relief.*] Coughing or exertion produces dyspnœa; a pain in chest; once had hæmoptysis; has lost much weight. *N.B. After treatment.* Dec., '99.—Has more than regained lost weight; has no cough or wheeze.

46. Mr. F. F., æt. 40, July 29, '98. Variety, spasmodic; duration, 7 years; severity, 3. [Eleven sittings; complete relief.*] Hay asthma all summer for 7 years; during last 12 months asthma constant; unable to do any kind of work. *N.B. Under treatment.* Oct., '99.—"Been grand, but sneezing just begun again." Oct., 1901.—"Been perfectly well, except for some eye irritation during last 14 days."

47. Mrs. C. W., æt. 49, Aug. 4, '98. Variety, bronchial; duration, 2 years; severity, 2, 3. [Twelve applications; complete relief.*] Asthma began with seven-week attack of bronchitis; for 2 or 3 hours asthma every night; shortness of breath constant during day; can always prognosticate attack of asthma from feeling in nose.

48. Miss S., æt. 28, Aug. 11, '98. Variety, spasmodic; duration, 3 to 4 years; severity, 1, 2. [Six applications; complete relief.*] Hay fever for some years previously. *N.B. After treatment.*—Asthma relieved; hay fever returned.

49. Mr. J. M. A. R., æt. 40, Aug. 12, '98. Variety, spasmodic; duration, years; severity, 1, 2. [Four applications; complete relief.] *N.B.*—Constant irritating cough, which was relieved by removal of uvula. Septal spur removed from right side. Septal spur right.

50. Mrs. M. A., æt. 48, Aug. 14, '98. Variety, spasmodic; duration, years; severity, 1, 2. [Four applications; complete relief.*] Suffers from diabetes; breathing bad at night; always needs 7 pillows. *N.B. After treatment.*—Sleeps well, naturally.

51. Mast. J. F., æt. 14, Aug. 15, '98. Variety, spasmodic; duration, 2 years; severity, 2, 3. [Thirty applications; complete freedom for months at a time.*] Asthma every night, frequently throughout the day. *N.B. Under treatment.*—Gradual but steady improvement.

52. Mr. C. S., æt. 55, Aug. 20, '98. Variety, spasmodic; duration, 30 years; severity, 3. [Two sittings; complete relief.*] Had pneumonia three times; heart weak; dyspnœa constant; very severe,

attacks frequently. *N.B. After treatment.*—No sign of asthma, except slight wheeze after long journey in '99. Small polypus on left not touched.

53. Mr. J. C., æt. 30, Aug. 26, '98. Variety, bronchial; duration, 1 year; severity, 1, 2. [Ten sittings; complete relief.*] Began with an attack of bronchitis which lasted 6 weeks; now wheezes from 1 to 4 a.m. *N.B. Under treatment.*—Improvement gradual.

54. Mrs. R., Aug. 27, '98. Variety, spasmodic; duration, 2 years; severity, 1. [Two sittings; complete relief till lost sight of.*] *N.B. After treatment.* Aug., '99.—Reported "breathing quite right now."

55. Mr. E. C. C., æt. 56, Sept. 6, '98. Variety, spasmodic; duration, 8 years; severity, 3. [Four sittings; greatly improved; lost sight of.*] Attack begins in afternoon; gets easier, but returns at midnight; no night free during last 3 years. *N.B. Under treatment.* Sept. 13.—Only one attack since. Oct. 11, '98.—Better than for years; can sleep now all night.

56. Mr. J. A. D., æt. 50, Sept. 7, '98. Variety, spasmodic; duration, since childhood; severity, 3+. [Eighteen sittings; great improvement.*] Has had pneumonia several times; twelve years ago was free for short time; asthma now constant; has had to sit up every night for 3 months. *N.B. Under treatment.* Oct. 28.—Very well, but some hay fever. Mar. 28, '99.—No bad night since. Feb. 2, 1900.—"Grand." Nov., 1901.—Had pneumonia; asthma returned. Jan., 1902.—Quite free of asthma.

57. Mr. W. L., æt. 48, Sept. 17, '98. Variety, bronchial; duration, 2 years; severity, 3+. [Three applications; complete relief.*] Began with bronchitis; hospital for 3 months; sent thence to consumptive sanatorium; left there in order to die at home. *N.B.*—First treatment had no effect; second stopped asthma completely; gained one stone in weight in first month; can now do manual labour.

58. Mr. G. M., æt. 40, Sept. 26, '98. Variety, spasmodic; duration, 4 years; severity, 2. [Four applications; complete relief.*] Began after hay fever.

59. Mr. E. H. K., æt. 23, Sept. 27, '98. Variety, spasmodic; duration, 6 years; severity, 2. [Eleven applications; complete relief.*] History of asthma in family; attack 2—4 a.m. every night; uses Himrod constantly.

60. Mr. W. S., æt. 45, Sept. 28, '98. Variety, spasmodic; duration, 5 years; severity, 3+. [Twelve applications; great improvement; lost sight of.*] "For last 12 months can hardly breathe or move." *N.B. Under treatment.* Sept. 30.—Marked improvement. Feb. 13, '99.—On the whole wonderfully well; able to do work, but has had attacks at times.

61. Mr. L. S., æt. 36, Sept. 28, '98. Variety, spasmodic; duration, 9 years; severity, 2. [Nine applications; temporary relief.*] Patient convinced asthma due to indigestion. *N.B.*—Always better after polypus removed. "Pleasure to live now." Polypi.

62. Miss H., æt. 33, Sept. 29, '98. Variety, spasmodic; duration, many years; severity, 2. [Ten applications; complete relief.*] Was free for 10 years; now bad every night for last 12 months. *N.B.*—Quite relieved after third treatment. Nov., '99.—Had one attack. Sept., 1901.—No asthma since.

63. Mr. W. A., æt. 42, Oct. 1, '98. Variety, spasmodic; duration, 10 months; severity, 3. [Nineteen sittings; complete relief.*] For some months intermittent; now constant. *N.B.*—Several times got quite well under treatment, but excess of alcohol caused return of asthma. June 14, '99.—Considers himself quite cured. May 24, 1901.—No asthma since.

64. Mr. R. T., æt. 24, Oct. 5, '98. Variety, spasmodic; duration, 6 months; severity, 1. [Six sittings; complete relief.*] *N.B.*—First noticed wheeze on walking uphill; now dyspnœa on any exertion; frequently gets attacks 2—3 a.m.

65. Mr. T. W., æt. 50, Oct. 5, '98. Variety, spasmodic; duration, 2 years; severity, 3. [Twenty-one sittings; marked relief.*] Asthma steadily increased in severity; now constant; specially severe at night; can get no sleep; "been given up for dead several times." *N.B.*—Temporary relief on removing polypi and treating septum; he eventually got well enough to do light work, and sleeps well. Many polypi.

66. Miss B., æt. 24, Oct. 10, '98. Variety, spasmodic; duration, 12 years; severity, 2. [Ten sittings; no permanent relief.*] Suffered from nasal obstruction and sneezing as long as can remember. *N.B.*—Got intermittent relief, but was obliged to return to country.

67. Mr. M. O. C., æt. 32, Oct. 12, '98. Variety, bronchial; duration, years; severity, 1, 2. [Five sittings; complete relief.*] Hurrying or exertion brings on cough and dyspnœa; frequent attacks at night.

68. Miss W., æt. 21, Oct. 15, '98. Variety, bronchial; duration, 5 years; severity, 3. [Thirteen sittings; complete relief.*] Very subject to bronchitis; has had asthma every night for 5 years, and frequently long and severe attacks. *N.B.*—Steadily improved under treatment; gained one stone in weight. Hypertrophic rhinitis.

69. Mr. B. B., æt. 50, Oct. 18, '98. Variety, spasmodic; duration, 8—9 years; severity, 2. [Eight sittings; complete relief.*] Attacks always preceded by sneezing. *N.B. After treatment.*—Occasional attacks of hay fever, but no asthma.

70. Mr. G. S., æt. 35, Oct. 19, '98. Variety, spasmodic; duration, 3 years; severity, 2. [Ten sittings; complete relief.*] Hay fever for 7 years. *N.B.*—Attacks always followed hay fever or indigestion.

71. Mr. J. G., æt. 73, Oct. 20, '98. Variety, cardiac (?); duration, 12 years; severity, 2. [Nine sittings; complete relief.*] Cannot walk many yards without attack; gets relief on resting; heart-

sounds very feeble; very marked arcus senilis. *N.B. After treatment.* Feb. 7, '99.—Walked 3 miles without any dyspnœa.

72. Mrs. L., Oct. 24, '98. Variety, bronchial; duration, 4 months; severity, 2. [Two sittings; improved; left district.*] Had attack 4 years previously; been very bad every night during last 4 months. *N.B. Under treatment.* Nov. 1, '98.—Greatly improved; no attack at night since.

73. Mr. E. R. H., æt. 27, Oct. 26, '98. Variety, spasmodic; duration, all his life; severity, 2. [Nine sittings; great improvement; left district.*] History of asthma in family. *N.B. Under treatment.* Feb. 16, '99.—Greatly improved; hardly any sign of asthma since.

74. Miss M., æt. 40, Oct. 31, '98. Variety, spasmodic; duration, 2 years; severity, 3. [Three sittings; complete relief.*] Asthma had been attributed to goitre, from which she had suffered 11 years; extreme dyspnœa on exertion, especially on lifting any weight.

75. Miss S., Oct. 31, '98. Variety, spasmodic; duration, all her life; severity, 3. [Three sittings; no relief; left for England.*] Has used Himrod powder at all hours of day and night for last 15 years.

76. Mr. L., Nov. 4, '98. Variety, spasmodic; duration, many years; severity, 2. Great relief; left district.

77. Mr. J. B., æt. 47, Nov. 4, '98. Variety, bronchial; duration, 4 years; severity, 2, 3. [Seven sittings; complete relief.*] Has had several attacks of pneumonia; now unable to do any active work; supposed to be tubercular.

78. Mrs. H., æt. 52, Nov. 7, '98. Variety, spasmodic; duration, 1½ years; severity, 2. [Eleven sittings; complete relief.*] Asthma had been attributed to large submaxillary growth, which she had for 12 years. *N.B.*—Obtained no relief from first 5 treatments.

79. Mr. J. S. B., æt. 48, Nov. 8, '98. Variety, bronchial; duration, 4 years; severity, 3. [Thirteen sittings; slight improvement.*] Began after bronchitis; now never quite free day or night; severe attacks frequently; can get no relief from medicines or inhalations. *N.B. After treatment.*—Attacks less frequent.

80. Mr. C. W., æt. 56, Nov. 15, '98. Variety, bronchial; duration, many years; severity, 2. [Eight sittings; complete relief.*] Began with bronchitis; has had much cough and phlegm for years.

81. Miss H., æt. 15, Nov. 17, '98. Variety, spasmodic; duration, many years; severity, 2. [Six sittings; complete relief.*] Rarely gets proper night's sleep on account of wheeze; much cough.

82. Mrs. B., Nov. 24, '98. Variety, spasmodic; duration, 3 weeks; severity, 2. [Three sittings; complete relief.*] Bad asthma every night on lying down.

83. Mr. C. M., æt. 55, Nov. 25, '98. Variety, spasmodic; dura-

tion, 2 years; severity, 2. [Three sittings; no relief.*] Has suffered from much sneezing for years.

84. Mrs. R., æt. 48, Nov. 28, '98. Variety, spasmodic; duration, 20 years; severity, 2. [Fourteen sittings; greatly relieved; rarely needs Himrod.*] Has used one tin of Himrod per week for the last 10 years.

85. Mr. E. P., æt. 59, Nov. 29, '98. Variety, spasmodic; duration, 4 months; severity, 2. [Ten sittings; no relief.*] *N.B.*—Got great relief from treatment at first. Very twisted septum, and impossible to reach posterior portion.

86. Mrs. T., æt. 30, Nov. 29, '98. Variety, bronchial; duration, 5 years; severity, 2. [Three sittings; great improvement till lost sight of.*] Began with bronchitis. *N.B. Under treatment.* Jan. 17, '99.—Much better.

87. Mr. E. T. H., æt. 43, Dec. 5, '98. Variety, spasmodic; duration, 9 months; severity, 3+. [Sixteen sittings; complete relief.*] Followed malarial fever; had tried all parts of Australia without obtaining relief; attributed trouble to malaria and indigestion. *N.B. Under treatment.*—First and second treatments produced no effect; derived instant relief after third cauterising.

88. Mrs. E., æt. 37, Dec. 5, '98. Variety, spasmodic; duration, 6 months; severity, 3. [Five sittings; complete relief.*] At first occasional attacks; now very severe.

89. Mr. M., æt. 17, Dec. 10, '98. Variety, spasmodic; duration, 6 weeks; severity, 2, 3. [Four sittings; great relief; lost sight of.*] Had suffered previously for 3 years, æt. 9—12. Perforating ulcer of cartilaginous septum.

90. Mrs. T., æt. 49, Dec. 11, '98. Variety, spasmodic; duration, 17 years; severity, 3+. [Twenty sittings; complete relief.*] Had been confined to house for 10 years; unable to walk; was lifted from bed to couch; any excitement induced severe spasm. *N.B.*—Immense relief after second treatment. On Dec. 26 was able to go for an all-day picnic, involving uphill walking, without any sign of asthma.

91. Mrs. P., æt. 33, Dec. 11, '98. Variety, spasmodic; duration, 8 years; severity, 2. [Seventeen sittings; great improvement.*] Always had severe attacks at menstrual period. *N.B. After treatment.*—Passed several periods without any asthma; needed uterine treatment.

92. Master L. B., æt. 9, Dec. 14, '98. Variety, spasmodic; duration, all his life; severity, 1. [Four sittings; complete relief Jan., 1902.*] Asthma with every cold. *N.B. After treatment.*—Has had several colds, but no asthma since.

93. Mrs. M., æt. 40, Dec. 15, '98. Variety, spasmodic; duration, 9 years; severity, 2. [Nineteen sittings; temporary relief on removing polypi.*] Began after attack of pleurisy; patient had always attributed asthma to indigestion. Many polypi.

94. Mr. J. B., æt. 45, Dec. 15, '98. Variety, spasmodic; dura-

tion, 20 years; severity, 1, 2. [Four sittings; temporary relief.*] Asthma with every change of weather. Polypi.

95. Mr. D. McL., æt. 40, Dec. 16, '98. Variety, spasmodic; duration, 4 years; severity, 2. [One sitting; complete relief Jan., 1902.*] Began after a cold; had inf. turbs. cauterised without relieving asthma; had been taking Pot. Iod. for 3 years; 4 days' freedom from asthma was longest period.

96. Mrs. K., æt. 53, Dec. 17, '98. Variety, spasmodic cardiac (?); duration, 20 years; severity, 3. [Twenty-six sittings; complete relief Nov., 1900.*] Bad for 4 years; then free for 6 years; then bad for 6 years, and free for 2 years; very bad during last 2 years; many doctors had told her that asthma was due to heart; used Himrod every hour.

97. Mr. J. R., Dec. 23, '98. Variety, spasmodic phthisis (?); duration, 9 years slight, 7 months very bad; severity, 3. [Six sittings; complete relief of asthma; cough continued.*] Slight attacks after having nose broken by horse kick; since August has had to sit up all night; lost 2 stone in weight; being treated for phthisis; no tub. bac. found in sputum.

98. Mr. W. B. R., æt. 33, Dec. 27, '98. Variety, spasmodic; duration, 3 months; severity, 2. [Eleven sittings; great relief.*] After influenza. *N.B. After treatment.*—Got complete relief of asthma till fresh attack of influenza, Mar., '99, when asthma again relieved by treatment.

99. Miss M. M., æt. 13, Dec. 29, '98. Variety, spasmodic; duration, years; severity, 1, 2. [Six sittings; great relief.*] Asthma with every cold; dyspnœa on any exertion. *N.B. After treatment.*—Sleeps well, but has asthma occasionally.

100. Mr. B. S., æt. 16, Dec. 29, '98. Variety, spasmodic; duration, 2 years; severity, 2. Eight sittings; complete relief.

101. Mr. P. M., æt. 35, Jan. 3, '99. Variety, spasmodic; duration, 23 years; severity, 3. [Fifteen sittings; great relief.*] Has used Himrod at all hours of day and night for last 14 years. *N.B. After treatment.*—Rarely needs Himrod.

102. Miss M., æt. 19, Jan. 5, '99. Variety, spasmodic; duration, 5 years; severity, 2. [Twenty-five sittings; attacks much less frequent after treatment.*] Deflection of septum to right, and hypertrophy of turbs. marked.

103. Mrs. S., æt. 47, Jan. 16, '99. Variety, spasmodic; duration, 21 years; severity, 3. [Nine sittings; complete relief.*] Began after sleeping in wet sheets; had interval of 9 years' freedom from asthma in North Queensland; asthma returned 12 months ago on coming to Brisbane; has not been to bed for last 3 months. *N.B. After treatment.*—Instant relief of asthma, but has some subsequent attacks.

104. Mr. J. L., æt. 35, Jan. 23, '99. Variety, bronchial phthisis; duration, 18 years; severity, 2. [Seven sittings; complete relief of asthma.*] Began in England; came to Queensland for sake of chest.

105. Mr. W. S. C., æt. 37, Jan. 27, '99. Variety, bronchial; duration, 5 years; severity, 3. [Thirty-one sittings; complete relief for 2½ years; asthma returned with attack of pneumonia; now improving under treatment.*] Came on with bronchitis after severe wetting in flood; now quite helpless; on four occasions relief obtained by bleeding when apparently asphyxiated. *N.B.*—No improvement till 19th treatment, when he obtained instant and complete relief.

106. Mrs. J. R., æt. 32, Jan. 27, '99. Variety, bronchial phthisis (?); duration, 2 years; severity, 3. [Eight sittings; complete relief.*] Came on with bronchitis; bad cough and much phlegm ever since; asthma every night and most days. *N.B. Under treatment.*—All chest trouble cleared up.

107. Mr. W. M., æt. 27, Jan. 30, '99. Variety, bronchial; duration, 24 years; severity, 2. [Eighteen sittings; complete relief.*] Fourteen years bad in England, 10 in Australia; asthma every night; very bad attack about every two months. Both sides.

108. Mr. B. G. A., æt. 40, Feb. 1, '99. Variety, spasmodic; duration, 9 years; severity, 3. [Ten sittings; complete relief.*] Had been taking large quantities of Iod. Pot. for years.

109. Mr. G. W. R., æt. 25, Feb. 4, '99. Variety, spasmodic phthisis (?); duration, 2 years; severity, 2. [Twelve sittings; great improvement.*] Has suffered from paroxysmal sneezing all his life; much cough and phlegm; is losing weight. *N.B. Under treatment.*—Sneezing relieved; asthmatic only at times; cough continues. Hypertrophy of mid. turb.

110. Mrs. G. F., Feb. 16, '99. Variety, spasmodic; duration, 7 years; severity, 1, 2. [Four sittings; complete relief.*] Has suffered for years from renal trouble; asthma every night from Jan. to June.

111. Mr. M. F., æt. 65, Feb. 20, '99. Variety, spasmodic; duration, 4 months; severity, 3. [Eight sittings; complete relief.*] Began suddenly; not had a free night since; dyspnœa on slightest exertion. *N.B. Under treatment.*—Relief complete at first till Mar. 21, '99. Relief again at once till Feb. 21, 1900; none since.

112. Mr. A. B., æt. 50, Feb. 20, '99. Variety, spasmodic cardiac; duration, 5 years; severity, 2. [Eight sittings; complete relief.*] Asthma preceded by heart symptoms, angina or palpitation.

113. Master J. McB., æt. 8, Feb. 23, '99. Variety, spasmodic; duration, 8 years; severity, 3. [Ten sittings; complete relief.*] Terrible cough and asthma since birth; walks like an old man, and has never been able to run. *N.B. Under treatment.*—Got complete relief after second treatment. Slight return April, 1900; none since.

114. Mr. P. C., æt. 24, Mar. 1, '99. Variety, spasmodic; duration, 15 years; severity, 2. [Three sittings; greatly relieved, but lost sight of.*] Attacks preceded by nasal irritation, *e. g.* after grooming a horse.

115. Mr. D. M., æt. 30, Mar. 6, '99. Variety, bronchial; duration, 2 years; severity, 2. [Thirteen sittings; complete relief.*] Came on with bronchitis; constant cold since; had been treated for phthisis. *N.B. Under treatment.*—Complete relief of asthma after Mar. 29, in spite of repeated "colds."

116. Mr. J. K., æt. 40, Mar. 6, '99. Variety, bronchial; duration, 1 year; severity, 3. [Nine sittings; complete relief.*] After pneumonia; never been free of asthma since; cannot walk across a room without severe dyspnœa; worse at night.

117. Mr. D. McL., æt. 51, Mar. 16, '99. Variety, bronchial; duration, 40 years; severity, 3. [Seven sittings; no relief.*] Family history of asthma; declined operation for removal of polypi. Polypi both sides.

118. Master J. D. McL., æt. 14, Mar. 25, '99. Variety, spasmodic; duration, 4 years; severity, 2. [Four sittings; greatly relieved; lost sight of.*] Son of Case 117; came on with cold.

119. Mrs. G. W., æt. 31, Mar. 28, '99. Variety, spasmodic; duration, 9 months; severity, 2. [Nineteen sittings; complete relief for three years; now under treatment again.*] Cold and change of weather bring on attack; sister died of phthisis. *N.B. After treatment.*—Got complete relief till after baby was born, Feb., 1902.

120. Mr. P. McC., April 5, '99. Variety, spasmodic; duration, years; severity, 2. [Two sittings; great relief; sleeping well; lost sight of.*] Asthma every night; fairly free by day.

121. Miss C., æt. 27, April 5, '99. Variety, bronchial (?); duration, 8 years; severity, 2. [Nine sittings; complete relief.*] Asthma nearly every night during summer; constant bronchial irritation.

122. Miss W., æt. 11, April 7, '99. Variety, spasmodic; duration, 3 years; severity, 2. [Eleven sittings; complete relief.*] Began after whooping-cough; mother had asthma at same age. *N.B. Under treatment.*—Asthma relieved at once; cough and night-sweats persisted for some time.

123. Mr. J. McF., April 10, '99. Variety, spasmodic; duration, 3 years; severity, 1. Four sittings; complete relief.

124. Mr. J. S., æt. 48, April 10, '99. Variety, spasmodic; duration, 20 years; severity, 3. [Six sittings; complete relief.*] Asthmatic attacks come on suddenly; depend on locality and emotions; once could not *sit* down at nights for 5 months; present attack came on after seeing friend in paroxysm of asthma; had not sat down for last 2 nights. *N.B.*—Obtained instant relief of paroxysm from first treatment.

125. Mr. J. J., æt. 40, April 12, '99. Variety, spasmodic; duration, 20 years; severity, 3. [Six sittings; complete relief.*] Obliged to use Himrod at least 3 times a day.

126. Mr. J. C. C., April 15, '99. Variety, spasmodic; duration, 2 to 3 years; severity, 2. [Four sittings; great relief; lost sight of.*] Complained of constant irritation in nose; asthma nearly every night.

127. Mr. E. B., æt. 20, April 22, '99. Variety, spasmodic; duration, 3 years; severity, 2. [Six sittings; complete relief.*] Asthma every night. *N.B.*—Obtained relief after first treatment.

128. Mr. G. H., æt. 67, April 24, '99. Variety, spasmodic; duration, 7 years; severity, 3+. [Six sittings; no relief; removed to North Queensland.*] Asthma constant; worse at night. *N.B. Under treatment.*—At first slept better; asthmatic paroxysm much modified.

129. Mr. J. M., æt. 28, April 25, '99. Variety, spasmodic; duration, 26 years; severity, 2. [Two sittings; complete relief.*] Wheeze constant while living at home in country; free when at sea-side.

130. Mr. A. F., æt. 35, April 25, '99. Variety, bronchial phthisis; duration, years; severity, 2. [Eleven sittings; asthma greatly relieved; went out West for sake of phthisis.*] Began with chest trouble. *N.B. After treatment.*—Attacks became less frequent and severe.

131. Mrs. W., æt. 35, April 26, '99. Variety, spasmodic; duration, 17 years; severity, 3. [Nine sittings; complete relief.*] Mother of Case 122; asthma very severe for 8 years in England; could obtain no relief from treatment there, so was sent to Queensland; change of climate afforded no relief; asthmatic attacks every night, and very severe all winter; attacks always preceded by irritation of nose. *N.B. After treatment.*—Slight attacks at night persisted for some considerable time. October, 1900.—Quite free of asthma all winter.

132. Mr. P. A. K., æt. 47, May 2, '99. Variety, spasmodic; duration, 3 years; severity, 2. [Six sittings; complete relief.*] Asthma always associated with indigestion; obliged to burn Himrod constantly.

133. Mr. W. B., æt. 19, May 3, '99. Variety, bronchial phthisis (?); duration, 2 years; severity, 2. [Eight sittings; complete relief.*] Began with a cold; much cough; hæmoptysis at times; had been treated for phthisis. *N.B. Under treatment.*—All chest trouble cleared up, with relief of asthma.

134. Mrs. C., æt. 63, May 4, '99. Variety, spasmodic; duration, 43 years; severity, 3. [Eleven sittings; considerable relief; went to live at sea-side.*] Burnt Himrod at all times day and night; better when at sea-side. *N.B.*—Got considerable relief from treatment, but was too sensitive and nervous to continue.

135. Mr. P. H. R., æt. 42, May 5, '99. Variety, spasmodic; duration, 25 years; severity, 2. [Ten sittings; great improvement.*] Began in England; became worse on coming to Australia. *N.B.*—Patient lives at great distance, and can only be seen at long intervals.

136. Mr. J. G. McD., æt. 68, May 10, '99. Variety, bronchial; duration, 5 years; severity, 2. [Nine sittings; considerable relief.*] Bronchitis and asthma began on coming to cold climate from tropics, where he had lived many years; asthma begins every

morning at 1 a.m. *N.B. After treatment.*—Sleeps well, but has dyspnœa on any exertion.

137. Mr. A. S., æt. 59, May 12, '99. Variety, spasmodic; duration, 14 years; severity, 2, 3. [Four sittings; temporary relief on removing polypi.*] Asthma constantly, and worse on change of weather. Many polypi.

138. Mr. A. H., æt. 30, May 12, '99. Variety, bronchial; duration, 6 weeks; severity, 2. [Two sittings; complete relief till lost sight of.*] Has had bad cough for years; for last 6 weeks has had to sit up every night. *N.B. Under treatment.* May 19.—"No sign of asthma since."

139. Mr. W. S., æt. 25, May 15, '99. Variety, spasmodic; duration, 15 years; severity, 2. [Six sittings; complete relief.*] Sneezes much; asthma always in winter; last summer also; never quite free during day, and worse at night.

140. Mr. F. W., May 15, '99. Variety, spasmodic; severity, 2. [Five sittings; great improvement; lost sight of.*] Acrid discharge from nose precedes attack.

141. Mr. A. D., æt. 35, May 25, '99. Variety, bronchial phthisis; duration, 9 months; severity, 2. [Seven sittings; asthma relieved; went out West for phthisis.*] Much cough, and occasional hæmoptysis for years.

142. Mr. W. K., æt. 45, June 1, '99. Variety, spasmodic; duration, 5 years; severity, 2. [Four sittings; complete relief.*] Always difficulty in breathing on any exertion; asthmatic attack every night; had been taking Pot. Iod. for years.

143. Mr. T. R. S., æt 40, June 2, '99. Variety, spasmodic; duration, 11 years; severity, 3. For some years attacks only occasionally in cold weather, on going out suddenly into the cold; now asthma constant day and night. *N.B.*—I could produce bronchial spasm by irritation of septum, which was instantly relieved by the application of cocaine. No relief.

144. Mr. L. W. B. H., æt. 15, June 5, '99. Variety, spasmodic; duration, 5 years; severity, 2. [Four sittings; complete relief.*] Attacks with every change of weather.

145. Mr. T. W., æt. 50, June 15, '99. Variety, spasmodic; duration, 30 years; severity, 2. [Twelve sittings; complete relief.*] Came on gradually; has not been to bed for 2 months. *N.B.*—After a few treatments got complete relief for 5 months, then fresh attack, and again complete relief for 8 months.

146. Mr. C. W. C., June 16, '99. Variety, bronchial; duration, 15 years; severity, 2. [Thirteen sittings; great relief.*] Had used Himrod every night for 15 years. *N.B.* June 30.—"Has not used Himrod for a week;" now uses Himrod occasionally, but never has bad attack.

147. Mr. J. W., æt. 35, June 20, '99. Variety, spasmodic; duration, 22 years; severity, 2, 3. [Seventeen sittings; complete relief.*] Since æt. 21 had never had proper night sleep; westerly

wind or impending rain always brought on attack. *Under treatment.*—Lost all tight breathing at night, until smell of "cobbler's peg" weed brought on sneezing and induced asthma.

148. Master C. McA., æt. 12, June 20, '99. Variety, spasmodic; duration, 3 weeks; severity, 2. [Five sittings; complete relief.*] Had persistent cough since bronchitis 4 months ago; asthma every night for last 3 weeks.

149. Mrs. D., æt. 30, June 24, '99. Variety, spasmodic; duration, —; severity, 1. [Eight sittings; complete relief.*] Complains of nasal obstruction. Hypertrophic rhinitis.

150. Mr. J. C. C., June 29, '99. Variety, spasmodic; duration, $2\frac{1}{2}$ years; severity, 1. [Two sittings; no record.*] Hay fever 12 years; asthma only in certain districts.

151. Miss E. B., æt. 13, July 7, '99. Variety, spasmodic; duration, 7 years; severity, 2. [Six sittings; complete relief.*] Attacks follow errors of diet; frequently had asthma day and night; not had a night free for two months.

152. Mr. W. R., July 17, '99. Variety, spasmodic; duration, 15 years; severity, 3. [Two sittings; complete relief till lost sight of.*] For last 3 months severe paroxysms day and night. *N.B.* July 21.—Complete relief.

153. Master G. R., æt. 5, July 19, '99. Variety, spasmodic; duration, $3\frac{1}{2}$ years; severity, 1, 2. [Three sittings; complete relief.*] Very frequently wheezing at night; several times a year had attacks day and night.

154. Mr. J. R., æt 48, July 24, '99. Variety, spasmodic; duration, 5 years; severity, 3+. [Thirty sittings; no relief of spasm.*] Has not had a fortnight free since it began; paroxysms now constant; great relief from freedom of the nose after removal of polypi. Complete nasal obstruction from polypi.

155. Mr. L. C., July 24, '99. Variety, bronchial; duration, 2 years; severity, 1. [Six sittings; complete relief of asthma, but not of sneezing.*] Much sneezing and bronchitis frequently.

156. Mr. C. U., æt. 50, July 24, '99. Variety, spasmodic; duration, many years; severity, 2. [Eight sittings; complete relief of asthma and sneezing.*] Sneezes much; nose always obstructed. Hypertrophic rhinitis.

157. Miss S., æt. 58, July 25, '99. Variety, bronchial; duration, 20 years; severity, 3+. [Fourteen sittings; complete relief.*] Had bronchitis for many years before asthma began; has tried all parts of Australia and Tasmania without obtaining relief; now quite helpless; had to be carried about.

158. Miss L., æt. 16, July 25, '99. Variety, spasmodic; duration, 12 months; severity, 2. [Six sittings; complete relief.*] Began after influenza and pneumonia.

159. Mr. W. H. C., July 27, '99. Variety, spasmodic cardiac (?); duration, 5 years; severity, 2. [Eight sittings; very great relief; left the country.*] Had heart trouble; always short of breath on

any exertion; great difficulty in going upstairs; severe attacks of asthma frequently. *N.B.*—After third treatment "went up two flights of stairs two steps at a time."

160. Mr. M. R., Aug. 10, '99. Variety, spasmodic; duration, 8 months; severity, 1. [Four sittings; complete relief.*] Preceded by much cough and irritation of the throat.

161. Mr. A. S., æt. 40, Aug. 10, '99. Variety, spasmodic; duration, 1 year; severity, 1, 2. [Nine sittings; no permanent relief.*] Came on after influenza; wakes 2 a.m. every morning. *N.B. After treatment.*—Relief obtained till Feb., 1900, when asthma returned at sea-side, and continued so long as he remained near the sea.

162. Mr. J. T., æt. 43, Aug. 12, '99. Variety, spasmodic; duration, 3 years; severity, 1. [Three sittings; complete relief April, 1902.*] Came on after bronchitis; bad asthma with every cold; short of breath at any time on exertion.

163. Mr. J. P., æt. 23, Aug. 16, '99. Variety, spasmodic; duration, since early childhood; severity, 3+. [Twelve sittings; complete relief.*] Sent from England on account of asthma; in England suffered one or two nights a week; since arrival in Queensland last April has been very bad every night and used Himrod constantly; "could ride a bicycle in England, but on no day since arrival could ride 100 yards on level ground."

164. Mr. C. W. T., æt. 60, Aug. 17, '99. Variety, spasmodic fibroid phthisis; duration, 2 years; severity, 2. [Fourteen sittings; greatly relieved; sleeps much better; only short of breath on exertion.*] Suffered from much cough and expectoration for 15 years.

165. Mr. H. J. D., æt. 37, Aug. 22, '99. Variety, bronchial; duration, 3 years; severity, 3. [Nineteen sittings; no relief.*] Has had several attacks of pneumonia; can only obtain relief from morphia injections.

166. Miss S., æt. 48, Aug. 24, '99. Variety, bronchial cardiac; duration, 10 years; severity, 3. [Twenty sittings; complete relief at first; now temporary relief after removal of polypus.*] Asthma bad day and night all winter; a little better in summer, but always breathless on any exertion. *After treatment.*—For two years experienced great improvement; no paroxysms; slept well; asthma then returned with development of polypus in nose. Polypus developed 2 years later.

167. Mr. T. M., Sept. 6, '99. Variety, spasmodic; severity, 2. [Four sittings; complete relief of all symptoms.*] Wakes three or four times a night with bad fits of coughing followed by asthma. *N.B.*—Touching septum with probe invariably induces bad fit of coughing.

168. Mr. M. M., Sept. 8, '99. Variety, spasmodic; duration, 18 months; severity, 2. [Five sittings; complete relief.*] Attack every morning early; shortness of breath during the day.

169. Miss K., æt. 30, Sept. 15, '99. Variety, spasmodic; duration, all her life; severity, 2. [Four sittings; complete relief (July 6, 1900).*] Marked periodicity; gradually gets worse till very bad, then better, then cycle repeated. *N.B.* Oct. 26, '99.—Has had bad cold and sneezing; no asthma.

170. Mr. F. B. C., æt. 17, Sept. 21, '99. Variety, spasmodic; duration, 14 years; severity, 1. [Eight sittings; complete relief until Jan. 2, 1900; lost sight of.*] Sometimes 3 months free; slightest cold brings on attack.

171. Mr. J. J. A., Sept. 22, '99. Variety, spasmodic; duration, 2 years; severity, 2. [Six sittings; complete relief.*] Wakes every night after 3 hours' sleep; then much cough followed by asthma. *N.B. Under treatment.*—Had bad cold in October, but no asthma.

172. Mr. W. S., æt. 24, Sept. 26, '99. Variety, spasmodic; severity, 1. [Five sittings; greatly improved.*] Obstruction in nose precedes attack; left for North Queensland.

173. Miss H., æt. 17, Sept. 29, '99. Variety, spasmodic; duration, since 10 months old; severity, 2, 3. [Three sittings; complete relief.*] At one time 3 or 4 attacks a day; now less frequently, but constant effort to breathe. *N.B.*—Instant relief on treatment.

174. Mr. H., æt. 48, Sept. 29, '99. Variety, spasmodic; duration, 10 years; severity, 2. [Five sittings; greatly improved; lost sight of.*] Often cannot work for weeks, rarely a night quite free; last seen on Oct. 26, when report "Very much better."

175. Miss M. K., æt. 11. Oct. 3, '99. Variety, spasmodic; duration, 2 years; severity, 1. [Twelve sittings; complete relief.*] Bad cough for 2 years; asthma comes on with every change of weather.

176. Mrs. W. D., Oct. 7, '99. Variety, bronchial; duration, 7 years; severity, 2. [Five sittings; complete relief.*] Asthma every morning early; rarely in daytime; very dry cough.

177. Mrs. W., Oct. 11, '99. Variety, bronchial; duration, 6 years; severity, 3. [Three sittings; lost sight of.*] Attack daily at 2 a.m., which continues all day. *N.B.*—Touching septum on either side induced spasm.

178. Mr. A. R. B., Oct. 20, '99. Variety, spasmodic; duration, 27 years; severity, 1. [Three sittings; great relief; left Brisbane.*] Asthma dependent on locality; very bad in Brisbane; certain smells produce an attack.

179. Miss G., æt. 20, Oct. 26, '99. Variety, bronchial phthisis; duration, 2 years; severity, 2. [Ten sittings; complete relief of asthma.*] Much cough; occasional hæmoptysis. *N.B.*—All wheezing ceased after Nov. 17.

180. Mr. C. D., æt. 47, Oct. 30, '99. Variety, spasmodic; duration, 8 years; severity, 3. [Ten sittings; great improvement.*] For last 9 days asthma constant day and night; suffers from albuminuria. *N.B.*—Got great relief from treatment; remained free till December; again relieved till August; another slight attack in September.

181. Mrs. M., æt. 33, Nov. 6, '99. Variety, bronchial; duration, 10 months; severity, 2. [Eight sittings; complete relief.*] Came on after a cold; much cough; losing weight; asthma at 3 a.m. every morning. *N.B.*—Instant relief from treatment; slight attack with a cold in December; none since.

182. Mrs. S., Nov. 10, '99. Variety, bronchial; duration, 10 months; severity, 2, 3. [Four sittings; complete relief.*] Asthma and severe coughing every morning at 2 a.m.; "smothering" all day. *N.B.*—Instant relief of bad nights; smothering during day continued for a time after treatment.

183. Master J. A., æt. 6, Nov. 11, '99. Variety, spasmodic; duration, 4 years; severity, 2. [Six sittings; complete relief.*] Came on after severe bronchitis; much cough and asthma every night. *N.B. Under treatment.*—Cough as well as asthma ceased.

184. Mr. J. S., Nov. 15, '99. Variety, bronchial phthisis; duration, 8 months; severity, 1, 2. [Four sittings; complete relief of asthma (Jan. 10, 1900).*] Began with a cold; much cough and phlegm every night, ending in asthmatic attack; occasional hæmoptysis. *N.B.* Dec. 8.—Can sleep now all night; then cough, but no wheeze.

185. Miss M. P., æt. 49, Nov. 15, '99. Variety, spasmodic; duration, 16 years; severity, 2. [Fifteen sittings; complete relief.*] Very severe attacks, sometimes lasting 3—4 weeks, at intervals of a few weeks. *N.B.*—Great relief from treatment at first; attacks again in May and July. Oct. 2, 1900.—No attack since Feb., 1900; complete relief since.

186. Mr. W. H. B., æt. 50, Nov. 18, '99. Variety, bronchial; duration, 1 year; severity, 2. [Eleven sittings; great improvement.*] Followed influenza and pneumonia; now uses Himrod every night. *After treatment.*—Sleeps well without Himrod; gets shortness of breath with colds, but no severe attacks.

187. Mr. E. W., æt. 32, Nov. 18, '99. Variety, spasmodic; duration, 5 years; severity, 2. [Seventeen sittings; great improvement.*] Had asthma as a child; then free till 5 years ago; since then been gradually getting worse; now an attack every night and often in daytime. *After treatment.*—Had slight attacks occasionally for some months. Jan., 1902.—"Only one slight attack since Aug., 1901."

188. Master D. F., æt. 14, Nov. 21, '99. Variety, spasmodic; duration, 10 years; severity, 2. [Eleven sittings; great improvement; lost sight of.*] Severe attacks frequently, and when so cannot lie down; unable to go to school. *After treatment.*—Instant relief; sleeps very well. Jan. 23, 1900.—Quite free since. Sept. 22, 1909.—Two slight attacks.

189. Master L. S., æt. 14, Nov. 22, '99. Variety, spasmodic; duration, 5 months; severity, 1. [Ten sittings; complete relief.*] Attacks following indiscretion in diet, or colds; frequently at night

without apparent cause. Deflection of septum to left, and hypertrophic rhinitis.

190. Mr. A. M., Nov. 23, '99. Variety, spasmodic; duration, 10 months; severity, 1. [Four sittings; great improvement.*] Butcher by trade; obliged to go to work very early; cold air produces cough and wheeze.

191. Miss L., Nov. 24, '99. Variety, bronchial; duration, 6 months; severity, 2. [Five sittings; complete relief of asthma.*] Asthma with much coughing every morning at 4 a.m.; attack on exertion at any time. *N.B.* Nov. 28, '99.—No wheeze since Sept. 4, 1900; bad cough last 3 months, but no asthma.

192. Master C. D. F., æt. 7, Nov. 24, '99. Variety, bronchial; duration, 2 years; severity, 1. [Four sittings; complete relief.*] Asthmatic attacks about once a month, when cannot lie down; always been mouth breather; removal of adenoids helped breathing, but did not prevent return of asthma; galvano-cautery to septum on left stopped wheeze.

193. Mr. A. E. J., æt. 29, Nov. 29, '99. Variety, spasmodic; duration, 5 years; severity, 1. [Four sittings; great improvement.*] Always bad in February and March; attacks then every night. *N.B.* Mar. 29, 1900.—"Sleeping only once interrupted."

194. Mrs. Z., æt. 40, Dec. 2, '99. Variety, spasmodic; duration, 5 years; severity, 2. [Eleven sittings; complete relief.*] Attack with every change of weather; cannot lie down; much cough. *N.B.* Feb., 1900.—Slight attack.

195. Mrs. M., æt. 50, Dec. 4, '99. Variety, bronchial; duration, 2 years; severity, 1. [Four sittings; greatly improved.*] Slight wheeze always; often had to sit up at night.

196. Mr. H. S., Dec. 10, '99. Variety, bronchial phthisis; duration, 2 years; severity, 2. [Twelve sittings; no improvement.*] Began in Africa; cough, much phlegm; occasional hæmoptysis; dust of work as bricklayer brings on attacks.

197. Mr. S. S., Dec. 14, '99. Variety, spasmodic; duration, 2 years; severity, 1. [Ten sittings; complete relief.*] Great sneezer; has to sit up with asthma about once a week. *After treatment.*—Asthma soon ceased, but sneezing persisted for some months. Hypertrophy of mid. turbs. very marked.

198. Master J. R., æt. 6½, Dec. 16, '99. Variety, bronchial; duration, 7 months: severity, 2. [Two sittings; lost sight of.*] Has had bronchitis several times; bad attack 7 months ago; breathing not free since. *N.B.* Dec 21, '99.—Greatly relieved.

199. Mr. T. C. N., Dec. 30, '99. Variety, spasmodic; duration, 4 years; severity, 1. [Six sittings; great improvement.*] Asthma only in certain localities; attacks preceded by headache and nasal oppression; complete relief for a time on removing part of mid. turb. and polypus. Polypus and hypertrophy of mid. turb.

200. Miss D., æt. 19, Jan. 6, 1900. Variety, bronchial; dura-

tion, years; severity, 1. [Two sittings; complete relief Feb., 1902.*] Asthma with bronchitis in winter.

201. Mr. L. C., Jan. 10, 1900. Variety, bronchial; duration, years; severity, 1. [Nine sittings; complete relief to Feb. 2, 1900, when lost sight of.*] Asthma almost every night, and two or three days a week during winter for some years; now occasionally in summer. *N.B.*—Relief at once on removing polypi and cauterising septum; return to North Queensland.

202. Mr. C. S., Jan. 11, 1900. Variety, bronchial; duration, 12 months; severity, 2. [Sixteen sittings; complete relief July 24, when he went to Germany.*] Came on with bronchitis; now always short of breath and severe attacks every week or 10 days. *Under treatment.*—Obtained relief on Jan. 18. Attack returned Feb. 12. Complete relief Feb. 18.

203. Miss N., æt. 18, Jan. 13, 1900. Variety, spasmodic; duration, 3 years; severity, 2. [Eleven sittings; marked relief.*] Has had trouble in nose for years; asthma gradually getting worse. *After treatment.*—Still gets attacks with sudden change of weather, but not severe. Atrophic rhinitis; ozæna; septal spur on left pressing against mid. turb.

204. Mr. J. I., Jan. 16, 1900. Variety, spasmodic; duration, 2 years; severity, 1. [Three sittings; lost sight of.*] Asthma in damp weather, otherwise once a month; got relief from being treated, but had to leave for north.

205. Mrs. G., æt. 50, Jan. 19, 1900. Variety, bronchial; duration, 6 years; severity, 2. [Five sittings; complete relief till lost sight of 2 months later.*] Came on after cold; asthma severe 2 or 3 nights a week; very bad all winter; returned in March to home in north.

206. Mr. H. S. C., Feb. 4, 1900. Variety, spasmodic; duration, 7 months; severity, 2. [Five sittings; complete relief.*] Great sneezer; asthma first in early morning, with much cough, then worse after meals. *N.B.*—Had no sign of asthma for 12 months after third treatment; then slight tightness which yielded at once.

207. Mr. W. M., æt. 16, Feb. 7, 1900. Variety, spasmodic; duration, 10 weeks; severity, 2. [Six sittings; complete relief.*] Very bad every night; much cough all night; no appetite. *N.B.* Feb. 14.—No asthma nor cough since. April 12.—"Splendid; never so well in life."

208. Mr. W. B., æt. 47, Feb. 8, 1900. Variety, spasmodic; duration, 4 years; severity, 3. [Twenty sittings; great improvement.*] Asthma every night from 12; can do no work at all by day. *Under treatment.*—Got free night occasionally up to June; then decided improvement; during last few months rarely troubled at night; still under treatment.

209. Mr. A. S. L., Feb. 8, 1900. Variety, spasmodic; duration, 3 years; severity, 2. [Two sittings; greatly improved; lost sight of; left for home in country.*] Came on after influenza;

asthma now at night and after meals. *N.B.* Feb. 21.—Breathing much better, and much better generally.

210. Mr. A. G. J., æt. 45, Feb. 12, 1900. Variety, spasmodic; duration, 1½ years; severity, 2. [Six sittings; complete relief.*] Bad every night in winter; attack at once if rides bicycle, or lifts weight. *N.B.* Mar. 30.—Had long ride on bicycle up hills without any wheeze.

211. Mr. E. M., Feb. 16, 1900. Variety, spasmodic; duration, 4 years; severity, 2, 3. [Four sittings; complete relief for 10 days under observation.*] Attacks at 3 a.m. every morning, often lost during the day; lived in N.S.W.; had to leave on Feb. 26.

212. Master E. T. W., æt. 14, Feb. 22, 1900. Variety, spasmodic; duration, 3 years; severity, 2, 3. [Twelve sittings; complete relief.*] Asthma nearly every night, sometimes all night and day; always dyspnœa on any exertion.

213. Mr. T. T., æt. 40, Feb. 14, 1900. Variety, spasmodic; duration, 18 years; severity, 1, 2. [Fourteen sittings; great improvement.*] Attacks last 2 to 3 days about every 3 weeks; sudden change always induces an attack. *After treatment.*—Got complete relief for months in spite of bad weather; but slight recurrence at intervals.

214. Mrs. H., æt. 29, Mar. 17, 1900. Variety, spasmodic; duration, years; severity, 1. [Six sittings; complete relief.*] Bad for 3 or 4 months a year. During Feb., Mar., and April, attack every night.

215. Mr. J. B., æt. 40, Mar. 17, 1900. Variety, spasmodic; duration, 24 years; severity, 2. [Two sittings; temporary relief on removing polypus.*] Rarely free; attack every 14 days; always dyspnœa when lying down. Polypus right.

216. Mr. A. A. S., Mar. 19, 1900. Variety, spasmodic cardiac phthisis; duration, 5 years; severity, 2. [Six sittings; great relief of spasm; cough and sweats continued.*] Nose always been obstructed; much cough; occasional hæmoptysis; mitral disease; bad asthma at least once a fortnight; has to sit in chair for nights.

217. Mr. W. R., Mar. 19, 1900. Variety, spasmodic; duration, 6 months; severity, 2. [Eleven sittings; complete relief of asthma.*] Asthma nearly every night; much cough and phlegm. *N.B.* Aug. 5.—Cough very worrying, but no asthma.

218. Mr. E. S., æt. 32, Mar. 22, 1900. Variety, spasmodic; duration, years; severity, 2. [Twelve sittings; considerable improvement.*] Great sneezer; asthma at first about once a month; now rarely free. *After treatment.*—Returns with cold or indigestion; no severe attacks.

219. Mrs. B., æt. 40, Mar. 26, 1900. Variety, spasmodic; duration, 14 years; severity, 2, 3. [Fourteen sittings; complete relief.*] After pneumonia; at first occasionally, now rarely free; often can hardly breathe or speak. *Under treatment.*—Improve-

ment only gradual for some months. Marked hypertrophy of mid. turb. on both sides.

220. Mr. J. H., Mar. 29, 1900. Variety, spasmodic; duration, 10 years; severity, 1, 2. [Thirteen sittings; sleeps better, but no marked improvement.*] Eight years free, until 6 months ago; apparently due to digestion. *After treatment.*—Was quite well till after eating Madagascar beans, when had very violent attack.

221. Mr. W. M., æt. 40, Mar. 29, 1900. Variety, spasmodic; duration, 4 years; severity, 2. [Four sittings; no improvement; left district.*] Suffered from severe sneezing for years.

222. Mr. W. L., April 7, 1900. Variety, spasmodic; duration, 20 years; severity, 3. [Two sittings; improved while under treatment; lost sight of April 20.*] Asthma very bad; last 9 years every night; often throughout day. Polypoid condition of mid. turb.

223. Mr. E. R., April 14, 1900. Variety, spasmodic; duration, years; severity, 1, 2. [Two sittings; lost sight of; returned to North Queensland.*] Came on after bronchitis; asthma only occasionally till last year; now every morning at 4 a.m.

224. Mr. J. McB., æt. 49, April 18, 1900. Variety, spasmodic; duration, 30 years; severity, 2, 3. [Eight sittings; complete relief.*] Asthma every night; very often during day. *N.B.* Feb., 1901.—Quite free of asthma, but still gets up at 2 a.m. for 10 minutes from habit, but has no dyspnœa nor cough now. Father of Case No. 113.

225. Mrs. B., æt. 52, April 30, 1900. Variety, bronchial; duration, 2 years; severity, 1. [Fourteen sittings; breathing greatly improved, but cough bad.*] After influenza; much cough and wheeze at night; chronic bronchitis all winter.

226. Mrs. J. M., æt. 33, May 3, 1900. Variety, spasmodic; duration, 25 years; severity, 3. [Eight sittings; complete relief.*] Asthma only occasionally for some years; now continuous since last Dec.; has not walked a hundred yards in 6 months; has been in one room for 2 months; sat in a chair for 16 days and nights. *N.B. After first treatment.*—Had best night for 4 months. May 5.—Went out for walk and had no sign of asthma; can now play tennis.

227. Mr. J. M., æt. 35, May 3, 1900. Variety, spasmodic; duration, 6 months; severity, 1. [Five sittings; temporary relief.*] Began in damp weather; complete relief temporarily on removing polypi, but still gets attack in damp weather. Polypi both sides.

228. Mr. J. T., æt. 44, May 7, 1900. Variety, spasmodic; duration, 12 months; severity, 1. [Seven sittings; complete relief.*] Comes on with cold; much irritating cough. *After treatment.*—Has had several colds, but no asthma.

229. Mr. J. T. B., May 7, 1900. Variety, bronchial; duration, 4 years; severity, 2. [Five sittings; greatly improved till lost sight of.*] After influenza; much cough; attacks at night and dyspnœa by day. *N.B.* May 21.—When he left for north cough much better; sleeping well, but short of breath on exertion.

230. Mr. D. S., æt. 32, May 7, 1900. Variety, spasmodic; duration, 7 years; severity, 2. [Eight sittings; complete relief.*] At first occasionally; since February not had a free night; sometimes bad by day. *N.B.* May 19, 1900.—"Never been so well in my life." May 28, 1900.—Had another attack; no sign since.

231. Mr. T. S., May 8, 1900. Variety, spasmodic; duration, 12 months; severity, 2. [One sitting; no record.*] Asthma at 1 a.m. every morning; very bad occasionally in the day. Polypus.

232. Miss T., æt. 27, May 8, 1900. Variety, bronchial phthisis; duration, 12 months; severity, 2. [Thirteen sittings; great improvement.*] After bronchitis; has now to sit up nearly all night; can do no work; even walking produces wheeze. *N.B.* May 28.—Not sat up at night since; can walk uphill. July 18, 1900.—First return of asthma. Dec. 11, 1900.—One slight attack since.

233. Miss McF., æt. 18, May 14, 1900. Variety, spasmodic; duration, 12 months; severity, 1. [Three sittings; relief complete on removing to dry climate.*] Nose completely obstructed; asthma on change of weather; experienced great relief on making nose free, but attacks occasionally. Polypus and hypertrophy of inf. turb.

234. Mr. T. G., May 14, 1900. Variety, spasmodic; duration, 4 years; severity, 3. [Six sittings; temporary relief; lost sight of.*] Uses Himrod every 2 or 3 hours day and night; great improvement on removing polypi. Polypi both sides.

235. Mrs. L., æt. 56, May 21, 1900. Variety, bronchial phthisis; duration, 3 years; severity, 2, 3. [Seven sittings; complete relief Aug. 27, 1900.*] After bronchitis; much cough; hæmoptysis; lost much weight; bad asthma every night. *N.B.* May 25.—Breathing better; does not wake till daylight. June 7.—Sleeping splendidly; all sign of spasm gone, although has had bad cold; can walk uphill with ease, although before treatment could not walk on level ground.

236. Mr. J. M., May 21, 1900. Variety, bronchial; duration, 12 months; severity, 2. [Nine sittings; great improvement; rarely gets any wheeze; never real attack.*] Constant colds; much cough and phlegm; asthma every night; has tried many localities and remedies in vain. *N.B.* June 1.—Great improvement; first relief ever experienced.

237. Mrs. W., æt. 64, May 23, 1900. Variety, bronchial; duration, years; severity, 2. [Two sittings; lost sight of.*] Asthma every night; much cough and phlegm day and night. *N.B.* May 25.—Sleeping better; breathing easier; not so much cough or phlegm.

238. Mr. M. M., æt. 48, May 24, 1900. Variety, ? spasmodic; duration, 7 years; severity, 3+. [Nine sittings; complete relief.*] Asthma comes on in cycles; gradually gets worse for nine days; then for 3 days and nights unable to sleep or move; then gradually better during 9 days; then cycle repeats; no remedies give relief; sneezes much *after* attack yields. *N.B.*—Immediately on cauterising right septum, right side of chest became free. May 25.—Right

side still free; left very tight; on cauterising left septum, left side of chest became free. May 26.—All tightness gone from chest; slept well; breathing easy. July 9.—Return to north quite well.

239. Mr. R. G., æt. 28, May 25, 1900. Variety, spasmodic; duration, 2 years; severity, 2. [Eight sittings; great improvement.*] Works in coal-mine; always bad in wet weather, and every night from 12 to 4 a.m. *N.B.*—Slept better after first treatment. Oct. 18.—Much better; sleeps well now.

240. Miss McD., æt. 19, June 6, 1900. Variety, spasmodic; duration, 18 years; severity, 1. [Eight sittings; temporary improvement; complete on removing to dry climate of west.*] Has had attacks since 10 months old; very bad at times with change of weather; removal of polypi gave relief, but polypi and asthma recurred. Many polypi.

241. Mr. A. R. A., æt. 40, June 6, 1900. Variety, spasmodic; duration, 4 to 5 years; severity, 1. [Eight sittings; considerable relief.*] On change of weather nose becomes obstructed; then asthma; removal of polypi gave great relief, still gets wheeze in wet weather. Polypi both sides.

242. Mr. F. C., æt. 34, June 11, 1900. Variety, bronchial; duration, 5 years; severity, 1. [Seven sittings; complete relief.*] After bronchitis; cough constant. Marked hypertrophy of inf. turbs.

243. Mrs. M., æt. 39, June 14, 1900. Variety, spasmodic; duration, years; severity, 1. [Eleven sittings; complete relief.*] Night air brings on wheeze. *After treatment.*—Free till 1 attack in September. None since.

244. Mr. A. F., æt. 59, June 15, 1900. Variety, spasmodic, ? cardiac; duration, 2 years; severity, 2. [Eleven sittings; great improvement.*] After influenza; sudden attack of asthma while digging in the garden; never quite free since; dyspnœa on any exertion; also from 11 to 1 p.m. and 4 to 5 a.m. *After treatment.*—Sleeps well, but still breathless on exertion. Septal spur on right.

245. Mr. G. W. S., June 25, 1900. Variety, bronchial phthisis; duration, 3 years; severity, 2, 3. [Two sittings; went out west for phthisis; lost sight of.*] Cough began 3 years ago; then asthma; wakes 2 or 3 times every night; has to sit up at least an hour each time; hæmoptysis at times. *N.B.* June 29.—Distinctly better; slept 5 hours without a break.

246. Mr. J. S., June 26, 1900. Variety, bronchial; duration, 1 year 10 months; severity, 2. [Two sittings; lost sight of.*] Bronchitis at first; "frightful cough;" much phlegm; bad asthma every morning at 2 a.m.; feels tightness all on the right side; physical signs of asthma on right side of chest only; touching right septum gave instant relief. *N.B.* June 27.—"What did you do to me yesterday? All my wheeze is gone, and I did not cough at all last night."

247. Mr. P. B., æt. 45, June 26, 1900. Variety, spasmodic; duration, 14 years; severity, 2. [Twelve sittings; no relief.*] Burns

powder every two hours at night; sometimes attack lasts for three days, when he can do nothing. *Under treatment.*—At first, interval at night became longer, and he was quite free by day; later breathing became as bad as ever.

248. Rev. A. N., æt. 37, June 26, 1900. Variety, spasmodic; duration, years; severity, 1, 2. [Five sittings; complete relief.*] Sneezes much; had nose cauterised in London without effect; wakes at 2 or 3 every morning, and has to sit up.

249. Mr. W. T. J., July 3, 1900. Variety, spasmodic; duration, years; severity, 1. [Three sittings; complete relief.*] Breathing always short; asthma with every cold. *N.B.* Jan. 13, 1901.—No return till caught cold, when breathing became tight, but no real attack; none since.

250. Mr. J. F., æt. 47, July 4, 1900. Variety, spasmodic; duration, 7 years; severity, 2. [Two sittings; complete relief; lost sight of.*] Dyspnœa on any exertion; during last 6 months asthma from 11 till 2 or 3 a.m. every night. Obtained instant relief from treatment.

251. Miss Y., æt. 22, July 4, 1900. Variety, spasmodic; duration, 14 years; severity, 1. [Ten sittings; great relief.*] Very bad attack from æt. 7—14; less frequent since; got relief at first without touching polypi; on asthma recurring, removal of polypi gave relief. Polypi both sides.

252. Miss C., July 6, 1900. Variety, spasmodic; duration, 5 years; severity, 2, 3. [Six sittings; complete relief.*] Constant attacks; worse at night; sometimes cannot lie down for weeks; instant relief after treatment; slight attack July 14. *N.B.* Dec. 10. —"Been splendid ever since."

253. Mrs. S., July 9, 1900. Variety, spasmodic cardiac (?); duration, 14 days; severity, 3. [Two sittings; lost sight of.*] Came on suddenly; up all night; can't dress herself; a feeling of faintness, then dyspnœa. *N.B.* July 13.—After first night slept well.

254. Mr. P. A., July 14, 1900. Variety, spasmodic; duration, 5 months; severity, 2. [Five sittings; greatly relieved.*] Great relief on removal of polypi. *N.B.* Jan. 18, 1900.—Much better altogether. Polypi.

255. Mr. J. A., æt. 42, July 16, 1900. Variety, spasmodic; duration, 38 years; severity, 2, 3. [Twenty sittings; very great improvement; now only rarely gets slight attacks.*] Had pneumonia twice; uses Himrod every night; gets very bad in smoke, dust, or cold winds. *N.B.*—Removed very large polypi from brother and nephew, who had no asthma. Very twisted septum.

256. Mrs. R., July 18, 1900. Variety, spasmodic; duration, 5 years; severity, 2. [One sitting; quite free when last heard of, Aug. 4.*] Very bad cough; severe asthma with every change of weather; sometimes laid up completely for a week; never free for more than 3 weeks; got relief of asthma without removing polypus. Polypus.

257. Mr. D. N., æt. 29, July 25, 1900. Variety, spasmodic; duration, 9 months; severity, 2. [Five sittings; complete relief when last seen, Sept. 8.*] Much cough; was off work for 5 months; tried different localities without benefit.

258. Mr. W. P. C., æt. 17, Aug. 6, 1900. Variety, spasmodic; duration, since childhood; severity, 1. [One sitting; lost sight of.*] Came on after "croup;" attacks for 2 or 3 days at a time, and usually wheezy till 10 a.m. every morning.

259. Mrs. B., Aug. 6, 1900. Variety, bronchial; duration, 2 years; severity, 3. [Three sittings; great improvement till lost sight of.*] Cough very severe; "asthma awful at nights; can't sleep; tired of all treatment and medicines." *N.B.* Aug. 13.—"Very good indeed, slept all night." Aug. 21.—Cough bad yesterday, but no asthma since."

260. Mr. R. A., æt. 38, Aug. 9, 1900. Variety, spasmodic; duration, 9 years; severity, 2. [Nine sittings; went back to north much improved; lost sight of.*] Can always sleep; very bad in day on any exertion. *Under treatment.*—Great improvement at first. Oct. 23.—Very well ever since. Dec.—One attack.

261. Mrs. D., Aug. 13, 1900. Variety, bronchial; duration, 2—3 years; severity, 2. [Four sittings; complete relief when lost sight of.*] Constantly taking cold; very bad asthma following; has to sit up all night. Aug. 13.—Terrible wheeze. Aug. 16.—Very well. Aug. 23.—None since.

262. Mr. C. E. S., Aug. 20, 1900. Variety, spasmodic; duration, 12 months; severity, 2. [Ten sittings; great improvement.*] Attacks frequently day and night. *Under treatment.*—Great improvement at first; relapsed, then improvement gradual; rarely gets attack now.

263. Mrs. B., æt. 54, Aug. 25, 1900. Variety, bronchial; duration, 4 years; severity, 2. [Three sittings; complete relief Sept. 25, 1901.*] Suffocating wheeze all winter; gradually getting worse.

264. Mr. P. H., æt. 39, Aug. 29, 1900. Variety, spasmodic; duration, years; severity, 2. [Six sittings; complete relief.*] Worse in winter; very bad at night; can never walk far.

265. Mr. T. C., æt. 50, Sept. 10, 1900. Variety, bronchial; duration, 18 months; severity, 2. [Six sittings; complete relief.*] Much cough and phlegm; breathing very bad until he can get up phlegm; cough and dyspnœa worse on lying down; had prolonged treatment for bronchitis without any relief; elongated uvula.

266. Mrs. E., æt. 45, Sept. 12, 1900. Variety, bronchial; duration, 18 years; severity, 3. [Fifteen sittings; some relief; still under treatment.*] Asthma every night and at any time on slight exertion; was much relieved by treatment till a severe attack of pneumonia, 1901.

267. Mr. H. R., æt. 24, Sept. 12, 1900. Variety, spasmodic; duration, 2 years; severity, 2, 3. [Seven sittings; complete relief.*]

Began after a wetting; cannot work; attack 2 a.m. every morning. *N.B.* Jan. 26, 1901.—No asthma since; sleeps well; better generally.

268. Mr. T. C., æt. 48, Sept. 19, 1900. Variety, bronchial fibroid phthisis; duration, 12 months; severity, 2. [Three sittings; went north; last report "better generally."*] Stonemason by trade; dyspnœa on any exertion.

269. Mr. S., æt. 52, Sept. 25, 1900. Variety, spasmodic; duration, 1 year; severity, 2. [Ten sittings; very great improvement; rarely wheezes; now can drink beer.*] First suddenly attacked at night; now every night on lying down; very often in day; beer or indiscretion in diet produces attack.

270. Mr. J. F., Oct. 3, 1900. Variety, bronchial; duration, 2 months; severity, 2. [Three sittings; complete relief.*] Came on with cold; for last six weeks has had to sit up in chair all night.

271. Mrs. W. J. G., æt. 35, Oct. 5, 1900. Variety, spasmodic; duration, 12 years; severity, 2. [Four sittings; complete relief.*] Had asthma æt. 17—23; now every night for at least 2 hours.

272. Mr. H. S., æt. 28, Oct. 20, 1900. Variety, bronchial; duration, all his life; severity, 2. [Eight sittings; great improvement.*] Very bad in Scotland; better at first in Queensland; now very bad during last 6 months. *N.B.* April 6.—Very well until slight attack 3 days ago. Aug. 3.—Very well; has had one attack since.

273. Mr. T. B., æt. 40, Oct. 24, 1900. Variety, spasmodic; duration, 12 months; severity, 2. [Four sittings; complete relief.*] Asthma at 2 a.m. every morning and on exertion.

274. Mr. C. H., Oct. 25, 1900. Variety, spasmodic; duration, 3 years; severity, 3. [Four sittings; complete relief till lost sight of.*] Asthma continues for last fortnight. *N.B.* Nov. 8.—Sleeping well; no dyspnœa.

275. Mr. G. A. S., Oct. 31, 1900. Variety, spasmodic; duration, 3½ years; severity, 2. [Ten sittings; great improvement till lost sight of.*] Asthma twice a night and on exertion; bad attacks during day sometimes. *N.B.* Nov. 13.—"Grand." Dec. 11.—Has had a bad cold, but no asthma; "never so free before." May 3.—Very well ever since. Aug. 22.—Another attack.

276. Mr. H. B., æt. 24, Oct. 31, 1900. Variety, spasmodic; duration, 4 years; severity, 1. [Five sittings; complete relief.*] Attacks occasionally by day; worse at night with much cough. Nov 17.—Breathing good; no asthma or cough at night. *N.B.* May 11, 1901.—One attack after typhoid fever.

277. Miss E. B., æt. 22, Nov. 7, 1900. Variety, spasmodic; duration, 3 to 4 years; severity, 1. [Five sittings; complete relief.*] Came on after pneumonia; now always has asthma after a cold.

278. Master B. L. S., æt. 11, Nov. 7, 1900. Variety, spasmodic; duration, 6 years; severity, 2. [Seven sittings; complete relief.*] Asthma every night; worse in summer; had had adenoids removed

three years previously in Canada, without any improvement of asthma. *N.B.* March, 1902.—No sign of asthma since.

279. Master J. M. W., æt. 6, Nov. 12, 1900. Variety, spasmodic; duration, 9 months; severity, 1, 2. [Two sittings; lost sight of.*] Much cough and wheeze at nights; has been gradually getting worse. *N.B.* Nov. 17.—"Better at night."

280. Mr. J. R., Nov. 13, 1900. Variety, bronchial; duration, 2 years; severity, 2. [Four sittings; complete relief till Nov. 30, when lost sight of.*] After bronchitis; now cough and asthma every night; removal of polypi gave relief. *N.B.* Nov. 30.—"No asthma since." Many polypi.

281. Mrs. J. G., æt. 60, Nov. 15, 1900. Variety, spasmodic; duration, 7 years; severity, 2. [Five sittings; much improved.*] Has required to use Himrod every night for last 4 years. *N.B.* Nov. 19.—Can sleep without Himrod.

282. Mr. J. McK., æt. 45, Nov. 16, 1900. Variety, bronchial; duration, 7 months; severity, 2. [Five sittings; complete relief.*] Had bronchitis for some months; carbolic acid inhalation set up an attack of asthma; now dyspnœa every night; steadily improved under treatment.

283. Mr. W. A. B., Nov. 26, 1900. Variety, spasmodic; duration, 10 years; severity, 2. [Five sittings; complete relief.*] Hay fever 14 years; asthmatic attack from 10 p.m. to 3 a.m., occasionally lasting for 2 days. *N.B.* Dec. 24.—Breathing and sleeping well.

284. Mr. H. P., Nov. 29, 1900. Variety, spasmodic; duration, 8 months; severity, 2. [Three sittings; relieved; lost sight of.*] Asthma every night; cannot sleep. *N.B.*—Cautery to right side of septum relieved right side of chest. Nov. 30.—Quite free on right side of chest; left side very tight. Dec. 5.—"Better."

285. Mr. S. B., æt. 19, Nov. 30, 1900. Variety, bronchial; duration, 3 weeks; severity, 2. [Two sittings; complete relief.*] Asthma from 2 a.m. till daylight every night.

286. Mr. W. C. C., Dec. 13, 1900. Variety, spasmodic; duration, 10 years; severity, 2. [Eight sittings; complete relief for 12 months; slight relapse; now again under treatment.*] Brother of Case No. 252; always has dyspnœa on exertion—very often at night; severe attacks frequently.

287. Mr. S. G. L., æt. 53, Dec. 11, 1900. Variety, spasmodic; duration, 2 years; severity, 2. [Five sittings; greatly relieved.*] Attack for 2 hours every night.

288. Mr. T. R. M., Dec. 22, 1900. Variety, spasmodic; duration, 18 months; severity, 2. [Six sittings; complete relief.*] Asthma from 2 or 3 a.m. every morning; gets relief from Himrod only.

289. Mr. W. E., Dec. 24, 1900. Variety, bronchial; duration, 6 months; severity, 2. [Five sittings; complete relief of asthma.*] Asthma from 2 a.m. every morning; dyspnœa on any exertion; severe cough with much phlegm; no relief from medicines. *N.B.* Feb. 22.—Cough bad in morning, but no asthma.

290. Mrs. S., Dec. 29, 1900. Variety, spasmodic; duration, 5 months; severity, 2. [Two sittings; better; lost sight of.*] Came on after bad confinement; wakes at same hour every night with asthma. *N.B.* Jan. 4, 1901.—Breathing much better, but still has cough.

291. Mr. A. C., Jan. 2, 1901. Variety, spasmodic; duration, 6 years; severity, 3. [Two sittings; no record.*] Bad every night; has to sit up from 12 midnight; asthma frequently in day. Polypi both sides.

292. Mr. S. J. H., Jan. 4, 1901. Variety, spasmodic; duration, 8 years; severity, 3. [Eight sittings; complete relief.*] Began with hay fever; very bad every night for last 5 years; bad attack with every cold. *N.B.* Jan. 7.—Feeling of bad cold in head, but no asthma. Jan. 12.—Influenza and slight dyspnœa. Jan. 22.—Breathing quite free; has had two attacks of bronchitis since, but no asthma.

293. Mr. W. M., Jan. 5, 1901. Variety, bronchial; duration, 8 years; severity, 2. [Six sittings; complete relief.*] Began with bronchitis; dyspnœa after food.

294. Mrs. M., Jan. 12, 1901. Variety, bronchial; duration, 18 months; severity, 1. [Four sittings; greatly improved; returned to Bush.*] Has had three bad attacks; constantly short of breath. *N.B.* Feb. 1.—"Splendid."

295. Miss R., æt. 28, Jan. 12, 1901. Variety, bronchial; duration, 14 years; severity, 1, 2. [Eight sittings; improved, but lost sight of.*] Asthma at 1 to 2 a.m.; not in daytime. *N.B.* Feb. 2.—Better. Mar. 9.—Had one attack in last 14 days; cough better.

296. Master C. H., æt. 16, Jan. 14, 1901. Variety, bronchial; duration, years; severity, 1, 2. [Fifteen sittings; complete relief.*] Bad attack whenever he gets cold; breathing always difficult. *N.B.*—Removed tonsils and part of turbs. Marked hypertrophic rhinitis.

297. Mr. S. A., Jan. 16, 1901. Variety, bronchial; duration, 12 months; severity, 2. [Eight sittings; complete relief.*] Asthma with much cough and phlegm every night; dyspnœa on exertion. *N.B.* Jan. 23.—Breathing very good; cough better. Mar. 14.—No asthma or cough; never wakes at night.

298. Miss G., æt. 14, Jan. 21, 1901. Variety, spasmodic; duration, 2 years; severity, 1. [Ten sittings; complete relief.*] Attacks occasionally; worse at night.

299. Master R. W., æt. 12, Jan. 23, 1901. Variety, bronchial; duration, 8 years; severity, 1. [Seven sittings; complete relief.*] Began after pneumonia; sneezes much; asthmatic attacks once in three weeks, which last about 12 hours.

300. Mr. T. F. R., æt. 18, Jan. 24, 1901. Variety, spasmodic; duration, since birth; severity, 1. [Four sittings; complete relief.*] Attacks occasionally with every cold.

301. Miss M., æt. 6, Jan. 25, 1901. Variety, spasmodic; dura-

tion, 12 months; severity, 2. [Ten sittings; complete relief.*] Asthma at night; very breathless on walking or running. *N.B.* Jan. 28.—Breathing better. Mar. 23.—Had one attack. May 7. —No asthma since.

302. Miss S., æt. 21, Feb. 7, 1901. Variety, spasmodic; duration, 3 months; severity, 2. [Six sittings; complete relief.*] Asthma every night; has to sit up from midnight till daylight. Sister of Case No. 100.

303. Miss H., æt. 30, Feb. 13, 1901. Variety, spasmodic; duration, 9 years; severity, 2. [Five sittings; great improvement; removed to country.*] Very bad during last 2 years.

304. Miss S., Feb. 15, 1901. Variety, spasmodic: duration, 14 years; severity, 2. [Seven sittings; complete relief.*] Asthma only slightly until last 9 months; very bad since; attack every 2 hours each night. *N.B.* Feb. 18.—Sleeping very well.

305. Mr. J. H., æt. 40, Feb. 19, 1901. Variety, bronchial phthisis (?); duration, 18 months; severity, 3+. [Seventeen sittings; great improvement.*] Began with cold while working on an ice cart; asthma all night and day; cannot sleep or walk; very severe cough with phlegm; had been considered a hopeless consumptive, but tubercle bacilli never found in sputum. *After treatment.*—Breathing greatly relieved at once. May 9.—Breathing well; can walk uphill; but cough still very bad.

306. Mrs. D. C., æt. 50, Mar. 1, 1901. Variety, spasmodic; duration, years; severity, 1, 2. [Nine sittings; complete relief.*] Wakes up gasping every morning; dyspnœa on any exertion.

307. Miss R., Mar. 4, 1901. Variety, spasmodic; duration, 20 years; severity, 3. [Seven sittings; great improvement till lost sight of.*] Asthma every night; has to smoke Warner's Safe Cure every hour; attacks frequently by day. *N.B.* Mar. 22.—"Great relief." April 11.—No asthma since. May 24.—Fresh attack.

308. Mr. W. G., æt. 33, Mar. 14, 1901. Variety, spasmodic; duration, 6 months; severity, 2. [Ten sittings; great improvement.*] Wakes every night at 2 a.m.; gets sleep after smoking cigarette; attacks sometimes in day. *N.B.* April 18.—Sleeping well. Aug. 1. —One attack, which came on immediately after last cauterising. June 25.—No asthma since. Aug. 23.—No attack since, but slight dyspnœa at times.

309. Mr. W. G., Mar. 15, 1901. Variety, bronchial: duration, 10 years; severity, 1, 2. [Seven sittings; complete relief.*] Sneezes much; bronchitis and asthma with every change of weather. *N.B. Under treatment.*—Wheeze soon stopped, but sneezing continued for a time.

310. Master J. C., æt. 13, Mar. 16, 1901. Variety, spasmodic; duration, 10 months; severity, 2. [Eight sittings; complete relief.*] Asthma every night; much cough and phlegm. *N.B.* April 25.—Decidedly better; sent to sea-side; returned quite well.

311. Mr. F. S., æt. 18, Mar. 16, 1901. Variety, spasmodic; dura-

tion, 2 years ; severity, 1. [Four sittings ; complete relief.*] Often gets cold in head ; then asthma at night. *N.B.*—Treated brother and sister for polypus without asthma.

312. Mrs. W., æt. 38, Mar. 16, 1901. Variety, spasmodic ; duration, 2 years ; severity, 1. [Twelve sittings ; considerable improvement.*] History of asthma in family.

313. Mrs. H., Mar. 17, 1901. Variety, spasmodic ; duration, 4 years ; severity, 1, 2. [Five sittings ; complete relief so far.*] Asthma during 4 months of each year—Feb., Mar., April, May ; then very bad every night. *N.B.* Mar. 24.—Two very slight attacks since 17. April 27.—No asthma since. May 19.—Quite free.

314. Mr. A. C. M., æt. 40, Mar. 22, 1901. Variety, spasmodic ; duration, 15 years ; severity, 2. [Twelve sittings ; complete relief.*] Very bad lately. *N.B.*—Very severe attack after first cautery. June 5 —Been very well. July 2.—Quite free.

315. Mr. J. A., April 6, 1901. Variety, spasmodic ; duration, 4 years ; severity, 1, 2. [Eight sittings ; great improvement.*] Never longer than a week or two without asthma. *N.B.*—Cautery always gives relief. Dec. 9.—" Very well."

316. Master R. H., æt. 14, April 8, 1901. Variety, spasmodic ; duration, 3 years ; severity, 2. [Five sittings ; complete relief.*] Asthma first came on after shooting in swamp ; during last 12 months very bad every night. *N.B.* Oct. 17.—No sign of asthma during all winter ; relapse of asthma after shooting in same swamp ; again relieved at once, and none since.

317. Mr. J. S., April 9, 1901. Variety, spasmodic ; duration, 3 months ; severity, 2. [Two sittings ; lost sight of.*] Asthma and bad cough every night. *After treatment.*—Breathing better, but still bad cough.

318. Mrs. McL., April 12, 1901. Variety, spasmodic ; duration, 12 months ; severity, 1. [Eight sittings ; complete relief.*] Comes on at night. *N.B.* May 21.—No asthma, but cough at night. Jan., 1902.—Very well.

319. Mrs. J., æt. 65, April 15, 1901. Variety, spasmodic cardiac (?) ; duration, 30 years ; severity, 2, 3. [Eight sittings ; temporary relief.*] Asthma on slightest exertion ; frequently very severe attacks ; attributed to worry and over-exertion ; removal of polypi and galvano-cautery to left gave relief. Many polypi.

320. Master J. S., æt. 6, April 17, 1901. Variety, spasmodic ; duration, 2 years ; severity, 1. [Four sittings ; complete relief.*] Wheezing and cough greatly improved after first treatment.

321. Mr E. F. H., æt. 33, April 20, 1901. Variety, spasmodic ; duration, since childhood ; severity, 2, 3. [Eight sittings ; great relief.*] Has had to sit up the whole of last 3 nights. *Under treatment.*—Greatly improved ; has had occasional attacks. June 17.—Feeling much better, but has had to use Himrod once or twice.

322. Hon. W. A., æt. 50, April 20, 1901. Variety, spasmodic cardiac; duration, 30 years; severity, 1, 2. [Two sittings; improvement.*] Has become worse during last 4 years; constant for last 3 months; walking from one room to another induces an attack; derived great relief from first treatment.

323. Capt. D., æt. 43, April 29, 1901. Variety, spasmodic; duration, 27 years; severity, 2. [Ten sittings; complete relief.*] Equally bad in all parts of the world, Arctic regions and tropics; very severe attacks at times; bad every night. *N.B. Under treatment.*—Very good fortnight; wonderfully better. June 17.—Has never been so well at this time of year.

324. Mrs. D. D., April 29, 1901. Variety, spasmodic; duration, years; severity, 2, 3. [Nine sittings; great temporary relief.*] Rarely a night free; dyspnœa on any exertion; bad attacks with every change of weather; derived immense relief from removal of polypi. Many polypi, which quickly recur.

325. Mr. H. B., April 29, 1901. Variety, spasmodic; duration, 7 years; severity, 3. [Five sittings; complete relief till lost sight of.*] Very bad at first; then easier till last December; much cough. *Under treatment.* May 14.—"Well till yesterday." May 30.—Quite well. June 13.—No wheezing since.

326. Mr. P. W., æt. 30, May 3, 1901. Variety, spasmodic; duration, 6 years; severity, 1. [Eight sittings; great improvement.*] Has only had three really bad attacks, but dyspnœa on exertion in the morning.

327. Master J. G. W., æt. 12, May 16, 1901. Variety, spasmodic; duration, 10½ years; severity, 1, 2. [Five sittings; complete relief.*] Began at æt. 18 months; breathing always difficult; attacks at times. *N.B.* June 13.—Has had bad cold, but no wheezing.

328. Mr. D. P., æt. 52, May 18, 1901. Variety, spasmodic; duration, 30 years; severity, 2. [One sitting; 1902 report, "Very seldom gets attack; visited Brisbane, but was not bad enough to need further treatment."*] "Lives on Himrod;" up two or three times every night; was obliged to return to north day after treatment.

329. Mr. J. O., May 18, 1901. Variety, spasmodic; duration, years; severity, 1, 2. [Six sittings; complete relief.*] Nearly every night from 12 to 3 a.m.; much cough at times. *N.B. Under treatment.* Nov. 2.—Has had influenza, but slept well at night.

330. Mr. J. W. O., æt. 40, May 23, 1901. Variety, spasmodic; duration, 8 years; severity, 1. [Three sittings; lost sight of.*] *N.B.* Cocaine applied to septum relieves breathing; under treatment (May 30) breathing better.

331. Mr. H. L., æt. 35, May 23, 1901. Variety, spasmodic; duration, 6 years; severity, 1. [Six sittings; great improvement; still under treatment.*] Wheeze every day; works in cold store. *N.B.*—Much better, but still under treatment; now bad only on Sundays.

332. Mr. T. M. M., æt. 60, June 24, 1901. Variety, spasmodic;

duration, years; severity, 2. [Eight sittings; great improvement.*] Very bad every night during the last 4 years; frequently in day for last 12 months. *N.B.* Sept. 20.—Very well till two nights ago. Report, March, 1902.—Very well now, but has had one or two attacks

333. Rev. J. K., æt. 44, June 26, 1901. Variety, spasmodic; duration, 7 years; severity, 2. [Six sittings; complete relief.*] Began in Scotland; not relieved on coming to Australia; usually every night; often completely laid up.

334. Mr. T. S., July 2, 1901. Variety, spasmodic; duration, 4 months; severity, 1, 2. [Five sittings; great improvement.*] Attack from 1 a.m. to 2 a.m. every morning; much cough. *N.B.* Nov. 23.—Sleeps well till daylight. Nov. 29.—One slight attack since.

335. Miss L., æt. 12, July 4, 1901. Variety, spasmodic; duration, 10 years; severity, 2. [Eight sittings; improvement.*] Attack nearly every night from 12 a.m.; bad in day when has cold. *After treatment.*—Only gets bad when has cold. *N.B.*—Mother has had asthma since birth of this child.

336. Master F., æt. 6, July 13, 1901. Variety, bronchial; duration, 2 years; severity, 2. [Four sittings; complete relief.*] Wheezing and very bad cough every night. *N.B.* Aug. 17.—Breathing much easier.

337. Mrs. D., æt. 60, July 16, 1901. Variety, bronchial; duration, 6 years; severity, 3+. [Ten sittings; some improvement.*] After bronchitis; has had no night's rest for 3 years, and attacks twice a day. *N.B.* July 20.—Somewhat better. Aug. 6.—Breathing decidedly easier. Aug. 17.—Attacks not so severe.

338. Mrs. McK., æt. 55, July 27, 1901. Variety, spasmodic; duration, 6 months; severity, 1, 2. [Three sittings; complete relief.*] "Spasm" in throat, and "choking" worse at night. *N.B. Under treatment* Aug. 8.—No choking since; has put on much weight.

339. Mr. T. I., æt. 53, July 27, 1901. Variety, spasmodic; duration, 8 years; severity, 1. [Nine sittings; complete relief.*] After influenza; cough on waking; then dyspnœa; bad attacks occasionally. *N.B.* Aug. 15.—No tightness. Two attacks of dyspnœa in November. Mar. 24, 1902.—Very well indeed.

340. Mr. L. McK., æt. 17, July 29, 1901. Variety, spasmodic; duration, since childhood; severity, 1. [Six sittings; complete relief.*] Often has wheeze in the morning; attacks of asthma about once a month. *N.B.*—Steadily improved under treatment. Aug. 27.—No sign of asthma.

341. Mrs. E. McM., æt. 41, July 31, 1901. Variety, spasmodic; duration, 12 years; severity, 2. [Five sittings; complete relief.*] At one time could not lie down for 12 months; an attack every Sunday without fail, and also at least two nights a week; always notices right nostril obstructed when asthmatic. *N.B. Under treatment.*—First Sunday quite free of asthma. Slight attack Sunday, Aug. 10.

342. Mr. W. H. P., æt. 20, Aug. 1, 1901. Variety, bronchial phthisis; duration, years; severity, 1, 2. [Seven sittings; removed to north; report Mar., 1902, "much better." *] Asthma at times; always much cough with phlegm; sometimes hæmoptysis; dyspnœa on any exertion.

343. Miss B., Aug. 2, 1900. Variety, bronchial: duration, 12 months; severity, 1. [Three sittings; lost sight of.*] Much cough for last 12 months; bad asthma at times, when has to sit up all night. *N.B.* Aug. 9.—Cough very bad, but breathing much better. Sept. 3.—Breathing tight on three mornings.

344. Mr. M. W., Aug. 5, 1901. Variety, spasmodic; duration, 3 years; severity, 2. [One sitting; no record; had to leave Brisbane next day.*] Very bad in England; now travelling to find relief.

345. Mr. T. McG., æt. 40, Aug. 14, 1901. Variety, spasmodic; duration, 2 years; severity, 3. [Twelve sittings; complete relief.*] Much cough, followed by feeling of "lump" in throat, which causes great dyspnœa; many times friends have thought him dead; patient was sent by a doctor to have supposed growth removed from larynx. *N.B.*—Sensation of "lump" instantly disappeared on my treating nasal septum; later he suffered from occasional attacks of dyspnœa, which he referred to chest; gained 12 pounds in a few months.

346. Mrs. M., Aug. 15, 1901. Variety, spasmodic; duration, 5 years; severity, 1. [Six sittings; complete relief.*] Asthma with every cold.

347. Mrs. L., æt. 29, Aug. 17, 1901. Variety, bronchial; duration, 12 months; severity, 1, 2. [Seven sittings; slight improvement.*] Bronchitis frequently, when always gets asthma.

348. Mr. H. W., æt. 18, Aug. 19, 1901. Variety, spasmodic; duration, 4 years; severity, 2. [Four sittings; complete relief.*] Bad attacks frequently on any exertion; cannot even walk fast; uses Himrod constantly. *N.B.* Aug. 24.—Has used Himrod once only since. Sept. 21.—No asthma since; can run and chop wood. Oct. 19.—No sign of asthma since.

349. Mrs. T., æt. 66, Aug. 19, 1901. Variety, spasmodic; duration, 8 years; severity, 2, 3. [Ten sittings; great improvement.*] Asthma for 2 weeks out of every 3, and every night at 1 a.m.; Great relief from removing polypi. Many polypi.

350. Mrs. S., æt. 39, Aug. 29, 1901. Variety, spasmodic; duration, 2 years; severity, 1. [Six sittings; complete relief.*] Bad cough at times; asthma occasionally; supposed to suffer from chronic phthisis. *N.B.* Nov. 22.—Much better generally; no asthma.

351. Mr. S. R., æt. 50, Aug. 29, 1901. Variety, spasmodic; duration, as long as can remember; severity, 3. [Eight sittings; some relief.*] Asthma now constant; "can't eat or sleep." *Under treatment.* Sept. 21.—Breathing better. Nov. 18.—"Feels 50 per cent. better."

352. Mr. G. R., Aug. 30, 1901. Variety, spasmodic; duration, years; severity, 2. [Five sittings; greatly improved; lost sight of.*] Much cough; had been treated for phthisis. *Under treatment.* Sept. 9.—"Very well till last night." Sept. 16.—"Very well since."

353. Mrs. C., æt. 37, Sept. 13, 1901. Variety, spasmodic; duration, 7 years; severity, 2. [Eight sittings; great improvement; still under treatment.*] Always worse at night; has had little sleep for last fortnight; bad in day if weather changeable. *Under treatment.*—Steady improvement.

354. Miss L., æt. 12, Sept. 23, 1901. Variety, spasmodic; duration, since birth; severity, 2. [Six sittings; complete relief March, 1902.*] Has always had bad cough. *N.B.* Sept. 30.—Breathing much better. Oct. 7.—Been quite well; no cough.

355. Mrs. H., æt. 70, Sept. 29, 1901. Variety, bronchial; duration, 3 months; severity, 1, 2. [Four sittings; improvement.*] Very bad attacks with cold winds.

356. Mr. J. E., æt. 26, Oct. 2, 1901. Variety, spasmodic; duration, 5 years; severity, 1, 2. [Three sittings; complete relief.*] Been gradually getting worse; attacks at least every week during last month both day and night; worse at night. *N.B.* Oct. 10.—Quite well since; first time he has slept well for a month.

357. Mr. J. G., Oct. 5, 1901. Variety, spasmodic; duration, 4 years; severity, 1. [Four sittings; great relief; still under treatment.*] Attacks frequently, when can't lie down. *N.B.*—Much better; has had one slight attack since treatment began; longest period free for 2 years.

358. Miss A. F., æt. 10, Oct. 6, 1901. Variety, spasmodic; duration, 6 months; severity, 1. [Two sittings; complete relief.*] Had first attack the night before whoop of whooping-cough appeared.

359. Mr. J. C., Oct. 8, 1901. Variety, spasmodic; duration, 11 years; severity, 2, 3. [Three sittings; great improvement; lost sight of.*] Attack every night at sundown; inhales smoke every 2 hours during night. *N.B.*—By touching nasal septum on left produced cough and irritation in throat exactly where tightness of breathing felt.

360. Mr. W. J. H., Oct. 10, 1901. Variety, spasmodic; duration, 8 years; severity, 1, 2. [Five sittings; complete relief.*] Has had asthma for a few weeks in the winter, but every night during last month. *N.B.* Oct. 23.—Better, but still some tightness. Nov. 29.—No asthma; sleeping well.

361. Mr. J. McM., Oct. 14, 1901. Variety, spasmodic; duration, 25 years; severity, 3. [Three sittings; slight improvement; left district.*] Asthma constant during last 3 years; very bad at night. *After treatment.* Oct. 19.—"Slept well last night."

362. Mr. G. D., æt. 50, Oct. 16, 1901. Variety, spasmodic; duration, 3 months; severity, 2, 3. [Five sittings; much improved;

still under treatment.*] Came on after a cold; much cough; dyspnœa on exertion or stooping; has to use Himrod three or four times a night. *N.B.* Oct. 21.—Can walk up a hill without tightness. Mar. 4.—Better in every way.

363. Mr. E. A. F., æt. 60, Oct. 19, 1901. Variety, spasmodic; duration, 30 years; severity, 1 till lately, now 2. [Seven sittings; complete relief.*] Began after packing new-mown hay, which brought on severe attack of hay fever; asthma now nearly every night, and frequently by day. *N.B.* Oct. 25.—Breathing wonderfully better. Nov. 4.—Slept well ever since. Dec. 6.—No sign of asthma.

364. Mr. C. P., æt. 43, Oct. 19, 1901. Variety, spasmodic; duration, 15 months; severity, 2. [Eight sittings; complete relief.*] Asthma at 4 a.m. every morning; continues at times during day; galvano-cautery to left side of septum made chest free instantly.

365. Mr. J. T., æt. 30, Oct. 20, 1901. Variety, bronchial; duration, 2 years; severity, 2. [Nine sittings; complete relief.*] Has had bronchitis for two years. *N.B.* Dec. 18.—"Very well; sleeps like a top; could run a mile."

366. Mr. T. H., æt. 47, Oct. 22, 1901. Variety, spasmodic; duration, 4 years; severity, 1. [Four sittings; complete relief to date.*] Asthmatic attack once or twice a year; present attack has lasted 3 weeks. *After treatment.*—Attack stopped instantly.

367. Mr. W. M., æt. 40, Oct. 23, 1901. Variety, spasmodic; duration, 5 months; severity, 1, 2. [Three sittings; complete relief.*] Cannot fill lungs day or night; dyspnœa on any exertion.

368. Mr. W. W. C., æt. 39, Oct. 29, 1901. Variety, spasmodic; duration, 9 years; severity, 2. [Five sittings; great improvement: still under treatment.*] Began after exposure in flood; attack lasting 13 days and nights; now every night from 1 a.m. till daylight. *After treatment.*—Now sleeping well; rarely an attack.

369. Mr. J. T. E., æt. 29, Nov. 4, 1901. Variety, spasmodic; duration, 10 years; severity, 2. [Seven sittings; great improvement; still under treatment.*] Much worse in the winter; certain smells—*e. g.* sealing-wax, resin, etc.—bring on attack at once.

370. Miss B., æt. 17, Nov. 12, 1901. Variety, spasmodic; duration, 10 months; severity, 3. [Five sittings; complete relief.*] Began suddenly on Jan. 24; not been free since; burns Himrod constantly. *N.B.* Nov. 15.—No asthma since. Nov. 22.—Quite well.

371. Miss W., æt. 11, Nov. 12, 1901. Variety, spasmodic; duration, 4 years; severity, 2. [Three sittings; complete relief.*] Began with a cold; worse at night; has sat up every night for last week. *N.B.* Nov. 19.—Breathing better; sleeping well. Dec. 7.—Very well; no sign of asthma.

372. Master S. F., æt. 9, Nov. 18, 1901. Variety, spasmodic; duration, 8 years; severity, 1, 2. [Nine sittings; great improvement; still under treatment.*] Attack with slightest cold; usually

lasts a week; improved steadily under treatment. *N.B.* Feb. 7.—Had never been so well in his life.

373. Mr. A. S. McL., æt. 21, Nov. 25, 1901. Variety, spasmodic; duration, 3 years; severity, 1. [Three sittings; no record; left for N.S.W.*] Attack about once in three weeks, preceded by irritation of nose; now suffering from an attack. *N.B.* Nov. 27.—Attack ceased.

374. Mr. R. J., Dec. 1, 1901. Variety, spasmodic; duration, 8 to 9 years; severity, 2. [Two sittings; complete relief (?); lost sight of.*] Last four weeks "can't sleep any night; can't eat; cares for nothing." *After treatment.* Dec. 9.—"Wants to sleep all day as well as all night;" no sign of asthma.

375. Mr. S. T., æt. 59, Dec. 5, 1901. Variety, spasmodic; duration, years; severity, 1. [Three sittings; complete relief till lost sight of.*] Very bad attacks at intervals; now suffering. *N.B.* Dec. 9.—Breathing much better. Dec. 13.—Quite well.

376. Master G. R., æt. 14, Dec. 6, 1901. Variety, spasmodic; duration, 12 years; severity, 2. [Three sittings; complete relief for 2 months, when left for N.S.W.; no record since.*] Attacks with every cold since æt. 2; during last 3 months has never been quite free, and bad at night. *N.B.*—Got great improvement at once from treatment.

377. Master H. H., æt. 14. Dec. 14, 1901. Variety, spasmodic; duration $12\frac{1}{2}$ years; severity, 3. [Seven sittings; complete relief at present; still under treatment.*] Never has a day quite free; has slept in sitting position for 6 years. *N.B. Under treatment.* Jan. 4, 1902.—Quite free; sleeping very well. Jan. 18.—Slight attack. Jan. 22.—No asthma since.

378. Mr. T. McM., æt. 40, Dec. 12, 1901. Variety, spasmodic; duration, 13 years; severity, 2, 3. [Eight sittings; complete relief.*] At first attacks lasting 7 to 14 days, at intervals of a few months; now more frequent; dyspnœa on any exertion; much cough and phlegm; has lost much weight; suspected phthisis. *After treatment.* Mar. 10, 1902.—All wheeze, cough, and phlegm gone; within 2 pounds of maximum weight; rode a bicycle 5 miles in 35 minutes without trouble.

379. Mrs. H., æt. 30, Jan. 7, 1902. Variety, spasmodic; duration, 9 years; severity, 1. [Three sittings; lost sight of; left for Victoria.*] Attacks about once a month; depend on locality. *After treatment.* Jan. 18.—Been staying at sea-side; quite free; usually sea-side brings on attack at once.

380. Mr. F. C., æt. 38, Jan. 8, 1902. Variety, spasmodic; duration, 2 years; severity, 2. [Five sittings; complete relief.*] Bad cough, followed by asthma, every night; suspected phthisis, but no tubercle bacilli found; works in cold storage rooms. *After treatment.* Jan. 20.—Quite free ever since in spite of continuous work.

381. Mrs. P., æt. 34, Jan. 22, 1902. Variety, spasmodic; dura-

tion, 6months; severity, 3. [Eight sittings; improving; still under treatment.*] *N.B. Later report.* June, 1902.—Asthma much improved, but tubercle bacilli found, and phthisis progressed. After an attack of pneumonia and pleurisy, asthma now every night; frequently in day; all signs of phthisis except tubercle bacilli in sputum; temperature, 4 p.m., varies from 99° to 101° daily.

382. Mr. W. E. R., æt. 30, Jan. 24, 1902. Variety, spasmodic; duration, all his life; severity, 2, 3. [Seven sittings; complete relief.*] Used to have long intervals, but gradually getting worse; now fairly constant; not been quite free for last 5 months; much cough and phlegm. *N.B. Under treatment.* Jan. 28.—Coughing, but phlegm much looser; no wheezing. Feb. 6.—Still coughing, but breathing splendid.

383. Mr. J. H., æt. 39, Feb. 1, 1902. Variety, spasmodic; duration, 2 years; severity, 2. [Four sittings; complete relief; still under observation.*] Began after bad cold; attack with every cold or change of weather; now every morning at 2.30 a.m.; suspected phthisis, but no tubercle bacilli found in sputum. *After treatment.* Feb. 8.—No tight breathing; "best week for 2 years." Mar. 8.—Has had bad cold, but no sign of asthma.

384. Mr. M. B., æt. 27, Feb. 3, 1902. Variety, spasmodic; duration, 2 years; severity, 1. [Six sittings; complete relief; still under treatment.*] Dyspnœa nearly every night; bad attacks occasionally. *N.B. Under treatment.* Feb. 24.—"Sleeping very well;" complains much of nasal obstruction, which was not relieved by reducing turbs. with ext. of supra-renal capsule, but instantly on touching septum on left with cocaine.

385. Mr. W. R., Feb. 8, 1902. Variety, bronchial; duration, 2 months; severity, 2. [Three sittings; improving; still under treatment.*] Had asthma 20 years ago; now bronchitis for last 2 months and asthma every night with much coughing. *N.B. Under treatment.* Feb. 12.—Breathing much better; cough very bad.

386. Master H. J., æt. 15, Feb. 12, 1902. Variety, spasmodic; duration, 3 years; severity, 1. [Three sittings; complete relief; still under treatment.*] Asthma in early morning, with bad cough. *N.B. Under treatment.* Feb. 24.—Wheezing gone; not coughing at all.

387. Mr. D. C., æt. 40, Feb. 13, 1902. Variety, spasmodic; duration, 4 years; severity, 2, 3. [Six sittings; improving; still under treatment. Later report, June, 1902, very marked improvement.*] Asthma constantly for last 9 days; has lost 3 stone in weight during last month. *N.B. Under treatment.* Feb. 24.—"Quite free till last night." Mar. 22.—Has had several attacks, but quite different now—more broken; can easily get up phlegm; attacks shorter, and recovery very rapid.

388. Mrs. B., Feb. 14, 1902. Variety, spasmodic; duration, 10 years; severity, 2, 3. [Four sittings; complete relief; still under observation.*] Has used Himrod every night and morning for last

6 months; very severe asthma if catches cold. *N.B. Under treatment.* Mar. 12.—Has not needed Himrod since first visit, and better generally.

389. Mr. A. W., æt. 24, Feb. 17, 1902. Variety, spasmodic; duration, 6 years; severity, 2. [Two sittings; complete relief; still under observation.*] Asthma came after severe wetting; now coughs for hours; then asthma every night. *N.B. Under treatment.* Feb. 28.—"Quite right ever since; sleeping grand;" no need to take medicine prescribed.

390. Miss B., æt. 26, Feb. 24, 1902. Variety, spasmodic; duration, 4 to 5 years; severity, 2. [Four sittings; complete relief.*] Severe asthma at times; once all summer; now nearly every night. *N.B. After treatment.* Mar. 13.—Quite free; sleeps well. Mar. 20. —Very well.

391. Master R. J., æt. 9, Feb. 28, 1902. Variety, spasmodic; duration, 5½ years; severity, 2, 3. [Four sittings; great improvement; still under treatment.*] Was perfectly well until he fell on an oil drum and broke the bridge of his nose; since then has had continual attacks of asthma and "bronchitis;" now has to sit up every night, and has dyspnœa by day. *N.B. After treatment.* Mar. 14.—No asthma since. Mar. 21.—Some wheezing one night on sudden change to cold. Mar. 27.—No sign of asthma since.

392. Mr. A. H. H., æt. 30, Feb. 28, 1902. Variety, spasmodic; duration, 3 years; severity, 2. [Six sittings; complete relief.*] Bronchial catarrh for last 3 years; dyspnœa at night, and on any exertion; has lost 1 stone in weight within the last month. Mar. 7. —Sleeping well; dyspnœa on walking uphill. Mar. 14.—"Can walk much better." Mar. 21.—Sleeping very well, and walking well. April 2.—Sleeps all night, and never short-winded now.

393. Mr. J. H., Mar. 3, 1902. Variety, bronchial; duration, 5 years; severity, 1. [Three sittings; complete relief; still under observation.*] Bad asthma 5 years ago, and at intervals since; very bad last 4 nights. *N.B. After treatment.* Mar. 6.—No trouble since. Mar. 14.—No return of asthma; cough gone.

394. Mr. A. B., Mar. 3, 1902. Variety, spasmodic; duration, 5 years; severity, 1, 2. [Five sittings; improving; still under treatment.*] Asthma about seven times a month and with every change of weather; sleeps in a canvas chair. *N.B. After treatment.* Mar. 27.—Very well lately.

395. Mr. T. S., æt. 50, Mar. 5, 1902. Variety, spasmodic; duration, 12 years; severity, 3. [Five sittings; complete relief; still under observation.*] Asthma every day and worse at night; feels every change of temperature; gardener by profession, but has been obliged to give up work. *N.B. Under treatment.* Mar. 11.— Had two good nights during week. Mar. 25.—Decidedly better; only one bad night in last fortnight. April 4.—Sleeps all night; has gone back to work.

396. Master G. G. F., æt. 9, Mar. 8, 1902. Variety, bronchial;

duration, 2 to 3 years; severity, 1. [Four sittings; complete relief; still under observation.*] Constantly catching cold, and then has asthma for about a fortnight; had hypertrophy of turbinate bodies and adenoids. *N.B. Under treatment.*—Breathing was greatly improved by cauterising the septum *before* operating for adenoids.

397. Mrs. B., Mar. 10, 1902. Variety, spasmodic; duration, 10 months; severity, 2. [Three sittings; improving; still under treatment.*] Began after influenza; bad asthma at night and on exertion; very weak and losing flesh rapidly; suspected phthisis. *N.B. Under treatment.* Mar. 18.—Sleeping better; no tubercle bacilli found in sputum. Mar. 25.—Sleeping well; eating better, but dyspnœa on any exertion.

398. Mr. W. G., Mar. 14, 1902. Variety, spasmodic; duration, 4 years; severity, 2. [Three sittings; great improvement; still under treatment.*] Asthma began with a cold; now every night; also on exertion; much cough. *N.B. Under treatment.* Mar. 15.—Cough better. Mar. 24.—Breathing quite free.

399. Mr. F. S., Mar. 15, 1902. Variety, spasmodic; duration, 17 years; severity, 3. [Three sittings; improving; still under treatment.*] Asthmatic attacks alternate with epileptic; bad every night; cannot lie down or do any kind of work. *N.B. Under treatment.* Mar. 18.—Has slept very well; can move about more easily. Mar. 19.—Still better.

400. Mr. J. C. S., æt. 23, Mar. 20, 1902. Variety, spasmodic; duration, 18 months; severity, 2, 3. [Three sittings; improving; still under treatment.*] Asthma came on suddenly; usually sits up all night; sometimes quite free for a week, but always worse immediately afterwards. *N.B. Under treatment.* Mar. 22.—Has had two good nights. April 7.—Been better; no tightness in breathing now till morning.

401. Mrs. F. H. S., Mar. 22, 1902. Variety, spasmodic; duration, since childhood; severity, 1. [Three sittings; relief; still under treatment.*] Bad asthma day and night for 2 or 3 weeks at time occasionally; has suffered much from hay fever; now has asthmatic attack. *N.B. Under treatment.* Mar. 29.—Asthmatic attack quite relieved.

402. Miss D., æt. 9, Mar. 25, 1902. Variety, spasmodic; duration, 3 years; severity, 3+. [Two sittings; improving; still under treatment.*] Has had pneumonia in both lungs; asthma much worse last 6 months; now cannot walk; very bad every night and nearly every day. *N.B. Under treatment.* Mar. 31.—Much better.

II.—*On Unilateral Atrophy of the Optic Nerve associated with hemiplegia of the opposite side.* By Leonard Guthrie, M.D., and Frederick E. Batten, M.D. *Read October* 10, 1902.

THE interest in the cases about to be recorded lies in the fact that the optic nerve was affected on one side only, viz. on the side of the lesion which caused the hemiplegia. That the condition is an uncommon one is certain, for but few cases have been reported, and after a careful search through the literature on the subject we have failed to find any case with a pathological description of the condition here described. We have come across many interesting cases of the same nature which have been reported clinically but not pathologically. One of our three cases terminated fatally, and at the autopsy occlusion of the left middle cerebral artery, causing atrophy of the convolutions supplied by that vessel, was found, together with atrophy of the left optic nerve. The atrophy of the nerve in this case can only be attributed to occlusion of the ophthalmic artery or one of its branches similar to the blockage of the middle cerebral artery. It is true that degeneration of the nerve-fibres in the optic chiasma and tracts was found, but this was secondary to affection of the optic nerve itself. We would emphasise this fact in order to differentiate the cases from those in which optic atrophy is due to damage of the *optic tract* on one side, for in such cases the atrophy must be bilateral. It may be mentioned that Sir William Gowers says, "Atrophy of one optic nerve may succeed softening, embolic or other, just as it succeeds hæmorrhage when the lesion has such a seat as to damage the nutrition of some part of the brain to which the optic fibres are related" (*Medical Ophthalmoscopy*, 1882, p. 130). But it is anatomically impossible that damage to one optic tract or its origin can produce complete atrophy of one optic nerve without affecting the other, unless, indeed, no decussation of the nerve-fibres takes place in the chiasma. To illustrate this distinction reference may be made to a case very fully reported by Dr. Shirres (*Studies from the Royal Victoria Hospital, Montreal*, vol. i, No. 2, January, 1902). This was a woman, the subject of congenital right hemiplegia, who died of pneumonia at the

age of 76. At the autopsy blockage of the left middle cerebral artery was found, with consequent atrophy of all the convolutions supplied by that vessel. The central ganglia were atrophied, including the left corpora quadrigemina and optic tract. The left optic nerve was smaller than the right, and certain bundles in the inferior portion were very markedly atrophied, whilst the right optic nerve also showed atrophy of its central bundles with general increase of glia tissue. Hence the neuritis was descending and bilateral in character, whereas in all our three cases there was no indication during life of damage to the optic nerve on the side opposite the lesion. In one it was shown by *post-mortem* examination that no such damage existed.

Case 1. *Girl æt.* 25. *Sudden onset right hemiplegia; sudden loss of vision in left eye; recurring fits, Jacksonian type; increasing coma; death.* Autopsy.—*Softening in distribution of the left middle cerebral artery; atrophy of left optic nerve; secondary degeneration of the spinal cord, optic chiasma, and optic tracts.*—The history of the case is as follows:—B. R., æt. 25, was admitted to the National Hospital under the care of Dr. Ormerod in May, 1899, and to him we are indebted for permission to publish this case. She had never been strong, but always anæmic, and subject to headaches. She had had no serious illness and neither rheumatism nor chorea. Her mother died of phthisis. Sixteen months previous to her admission to the hospital the patient had a fit during the night; she had felt quite well the day before. After the first fit she could not speak, though she could understand anything that was said to her. She was paralysed down the right side and had lost the sight of the left eye. It could not be ascertained with certainty whether the paralysis came on immediately after the fit or took some time to develop. The leg and face improved considerably, and after twelve months she could stand and walk, but there had been little improvement in the condition of the right arm.

Three months later she had another fit, and a third six weeks before admission to the hospital. During these fits the paralysed side was drawn up, but the other side did not move. For the last four months she had been troubled with retching. On admission to the hospital she was rather anæmic. Her intelligence was fairly bright. As regards speech, she understood easy sentences, but could not understand everything—*e. g.* when asked to shut her eyes and

open her mouth she did so, but when asked to touch the doctor's hand she touched her face. Articulation was almost unintelligible, and, as only a few words were occasionally made out, it was impossible to say whether she used correct words or not. Shown a written question, "Are you a hundred years old?" she seemed by her amused expression and laugh to appreciate the absurdity of it. She could not understand the written command, "Shut your eyes." Shown some letters, she recognised B, D, and Z, but called E, I. She recognised 5 and other numerals.

Sight.—The vision of the right eye seemed normal, the left eye was absolutely blind, and she could not tell light from darkness. There was no hemianopia in the right eye. *Discs.*—The right was normal; the left disc was very white and flat and the margin sharp, suggesting to Mr. Gunn, who saw the patient, the appearance met with in cases of thrombosis or hæmorrhage into the nerve-sheath. Further allusion will be made to this important observation by Mr. Gunn.

Hearing.—The ticking of a watch could be heard at one inch from the left ear and six inches from the right. She had been deaf, however, in the left ear all her life.

Cranial nerves.—Very slight external strabismus was present in the right eye. Movements of the eyes were normal, and there was no nystagmus. The right pupil reacted to light and on convergence. There was no reaction to light in the left pupil, but it gave a consensual reaction. Tongue deviated a little to the right. There was marked weakness of the lower part of the face on the right side. There was slight power of movement in the right shoulder, but none in the fingers. There was considerable rigidity of the right arm and leg, and she could walk with difficulty.

Sensation.—There was right hemianæsthesia.

Reflexes.—The elbow- and wrist-jerks were normal, but more active on the right side than on the left. The knee-jerk on the right side was greater than on the left. Ankle-clonus and extensor plantar response were present on the right side. A flexor response was present on the left. Tenderness to pressure on the head was more marked on the left side than on the right. The sp. gr. of the urine was 1018. It contained no albumen.

She vomited on June 11 and 12, and her headache was very severe on the night of June 12. The headache began behind the right ear, and extended later to the left frontal region.

PLATE I.

Illustrating Drs. Guthrie and Batten's paper on Unilateral Atrophy of the Optic Nerve associated with Hemiplegia of the Opposite Side.

Fig. 1.—Lateral view of the left hemisphere of the brain from Case 1, showing the area of softening involved in the blocking of the middle cerebral artery.

Fig. 2.—Horizontal section through the brain showing the area of softening on the left side, involving the cortex, the subcortex, the internal capsule, the lenticular nucleus, the outer part of the corpus striatum, and the optic thalamus. The general shrinking of the whole of the left side is well seen.

Fig. 1.

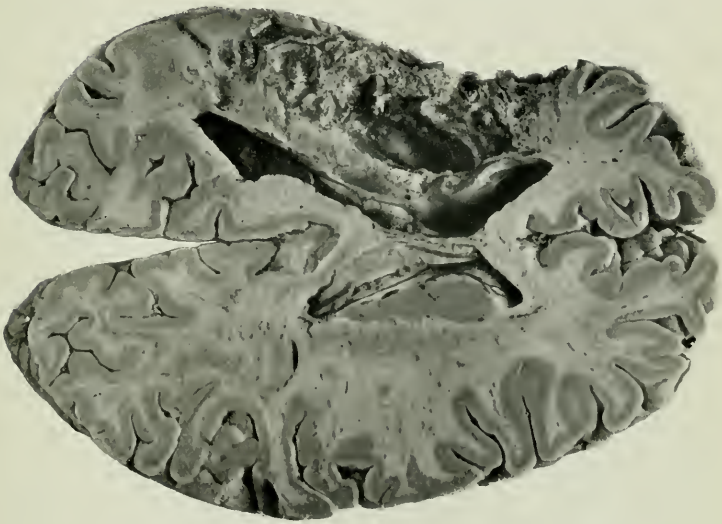

Fig. 2.

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On June 19 she had another fit, in which the head and eyes were turned to the right. There was no aura. The right arm became weaker after the fit.

On June 21 she was drowsy and complained of pain in the left ear.

On June 23 she complained of sharp, severe pain in the right arm and leg.

On June 26 she had two slight epileptic attacks at breakfast.

On June 28, as she was getting into bed, she turned very pale, felt giddy, and had slight twitching of the left hand.

On July 5 the headache was very severe on the left side.

August 27.—During the next five weeks she became very much worse. She was dull and heavy, and lay in a semi-comatose state and could not say a word. She passed her water unconsciously during the night. She occasionally put her hand up to the left side of her head. There was no vomiting during the last few days of life, but she had had some difficulty in swallowing. The patient gradually passed into a deeper coma, and died on September 1. Temperature, which was subnormal until thirty-six hours before death, rose to 110° at the time of death.

Autopsy performed four hours after death. Body well nourished. Skull natural. Dura mater and sinuses natural.

Brain.—After removal of the dura mater very marked atrophy of the left hemisphere was apparent; there was considerable compensatory œdema in the pia-arachnoid membrane over this side, and the convolutions were shrunken and firm; some œdema was also present on the right side. Situated around the left Sylvian fissure, involving the first temporo-sphenoidal and the lower portion of the third frontal, ascending frontal, and ascending parietal convolutions, was a space filled up with membrane that appeared in the same œdematous condition as the pia-arachnoid membrane above described. The convolutions of the occipital lobe were not so wasted as those of the parietal and frontal region. The left middle cerebral artery was hard and thrombosed.

The limits of the area of softening are well seen in the accompanying photograph (Plate I, figs. 1, 2). On section through the brain the difference in size between the two hemispheres was very marked, and the destruction of brain tissue on the left side was very extensive, involving the basal ganglia, the optic thalamus, the lenticular nucleus, and the outer part of the corpus striatum. The whole of the

structures involved were replaced by a kind of fine membranous material, the meshes of which were filled with fluid. Around the margins there was a good deal of broken-down and softened brain tissue. The corpora quadrigemina were of equal size and of normal appearance.

The left optic nerve appeared atrophied as compared with the right; but on measurement the cross-sections of the two nerves were found to be in each case 2 mm. × 3·5 mm.

The cerebellum and pons appeared normal.

Spinal cord appeared normal.

Thorax.—The pleuræ were adherent at one or two spots.

Lungs.—At the base of the left lung was an old calcareous mass measuring about 1·5 cm. in diameter, and there was also a small mass at the root of the same lung.

Heart.—Valves normal; no evidence of any endocarditis either old or recent; a few scattered petechiæ over the surface of the heart.

Abdominal organs normal.

Microscopical examination.—As the principal interest in the case centres in the condition of the optic nerves and chiasma, it may be well to describe the condition found in these structures first.

Marchi method.—Sections of the optic nerve, chiasma, and optic tracts cut in a horizontal direction showed that the left optic nerve was completely degenerated up to its junction with the chiasma. At this point the degeneration became scattered, evidently following the course of the fibres of the left optic nerve. The majority of these crossed the chiasma into the right optic tract, but others passed up into the left optic tract (*see* Plate II, fig. 2); the ultimate distribution of these fibres was not traced. No degenerated fibres could be found in the right optic nerve.

Transverse sections of the left optic nerve showed complete degeneration, with shrinking of the nerve and the formation of numerous spaces between the bundles of fibre.

Transverse section of the right optic nerve showed no degeneration.

Weigert-Pal method.—Horizontal sections of the optic chiasma stained by the Weigert-Pal method showed marked degeneration of the left optic nerve, and this degeneration could be traced up into the left optic tract, and also across the chiasma into the right optic tract. No degeneration could be seen in the right optic nerve (Plate II, fig. 1).

Pons, medulla, and spinal cord.—With regard to the

PLATE II.

Illustrating Drs. Guthrie and Batten's paper on Unilateral Atrophy of the Optic Nerve associated with Hemiplegia of the Opposite Side.

Fig. 1.—Photograph of a horizontal section through the optic tracts, chiasma, and nerves, stained by the Marchi-Weigert-Pal method, showing complete degeneration of the left optic nerve. The right optic nerve appears normal. In the chiasma and optic tracts the degeneration is more scattered and less clearly defined owing to the mingling of the degenerate and undegenerate fibres coming from the opposite optic nerves.

Fig. 2.—Drawing of horizontal section through the optic nerves, chiasma, and optic tracts stained by the Marchi method, showing complete degeneration of the left optic nerve with degeneration in the chiasma and both optic tracts. The right optic nerve appears normal.

Fig. 1.

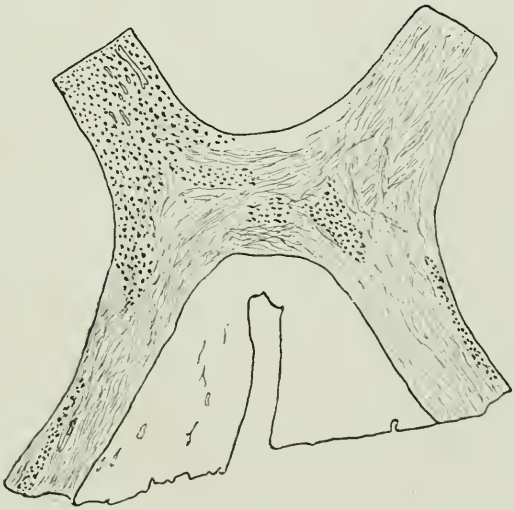

Fig. 2.

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pyramidal system, there was well-marked degeneration in the left pyramid above the decussation, and the whole of the left side of the pons appeared to be smaller than the right. This shrinking seemed to be almost entirely due to defect in the pyramidal fibres, although possibly the fillet on the left side did not appear quite so large or quite so well stained as that on the right. Below the decussation the degeneration could be traced down in the right pyramid to the sacral region. The degeneration of the direct pyramidal tract could be seen in the upper cervical region, but was no longer present at the level of the fifth cervical. (Hence it would appear that there was a nearly complete decussation of the pyramids.) The triangular bundle known as "Helweg's bundle" was present in the upper cervical region, and the degenerated ventro-lateral pyramidal tract was well seen.*

Case 2. *Right-sided hemiparesis with atrophy of the left optic nerve; complete recovery except for persistent pallor of disc.*—George S., æt. 12, a delicate-looking boy, attended the Hospital for Paralysis, Regent's Park, on April 30, 1901.

History.—He was quite well until October, 1900, when he fractured his left femur, and was admitted to St. Monica's Home on that account under the care of Dr. Winslow Hall, who kindly sent the following notes:

"January 22, 1901.—Plaster-of-Paris case removed; still callus round site of fracture.

"January 28.—To get up, and use crutches.

"February 7.—Complained of pain in lower third of left femur; rise of temperature at night; belladonna and glycerine applied.

"February 12.—Pain is now localised at a spot about one inch square on inner surface of thigh about the middle of the femur. No redness, no tenderness, and no swelling. Temperature lower.

"February 20.—Allowed to get up again.

"March 2.—Discharged cured."

Dr. Hall adds, "The febrile attack was a mystery to me throughout; there were no local signs to account for it, and the heart was unaffected. I called it rheumatism in default of a better diagnosis. The temperature, so far as I remember, was decidedly 'spiky;' there were no arthritic symptoms."

* See *Brain*, vol. xxiv, p. 492.

The patient came under the care of one of us (L. G.) on April 30, 1901, complaining of loss of power on the right side. He stated that in March, 1901, after being discharged from St. Monica's Home, he experienced numbness, tingling, and weakness of the right upper extremity. The two outer fingers of the right hand were chiefly affected. His handwriting became shaky, and he had difficulty in feeding himself owing to trembling of the right upper limb. About the same time he complained of numbness of the outer side of the right thigh and leg, and weakness of the whole limb. He was observed to drag it after him while walking, and the fore-part of his boot became quickly worn out. All these symptoms were gradual in onset and progressive in severity. When examined on April 30 there was weakness, incoordination, exaggerated reflexes, and marked intention tremor in the right upper extremity. The right thigh was somewhat wasted as compared with the left. The right knee-jerk was exaggerated, ankle-clonus was present, and the plantar reflex was markedly extensor in type. The right great toe gave a typical "Babinski" response, in striking contrast to the flexor reaction obtained on the other side.

Sensation was unimpaired; the gait was hemiplegic in character; there was no facial paralysis. The boy was somewhat anæmic, and a faint presystolic bruit was audible inside the cardiac apex-beat, but no other sign of cardiac disease was present.

Eyes.—Excursions normal. Pupils equal, somewhat dilated; reaction normal. Veins of left optic disc engorged, but disc not swollen. Right disc normal.

On May 21, general condition as before, but left optic disc hazy in outline, especially on inner side. Veins engorged. Vision not tested. He complained of temporary aphasia lasting a few minutes after watching a game of cricket.

June 18.—Vision very defective in left eye; could not count fingers at twelve inches. Left disc was now pale and the margins clear.

June 27.—Dr. Rayner Batten noted, "Left optic disc pale and excavated; cribriform plate exposed at bottom of excavation; disc not filled in nor opaque. No blurring of vessels nor of margin of optic disc. Well-marked central scotoma for white (no colour-perception). Retina in macular region and up to optic disc showed signs of disturbance and a number of fine white dots. Veins somewhat large, dark, and tense. No

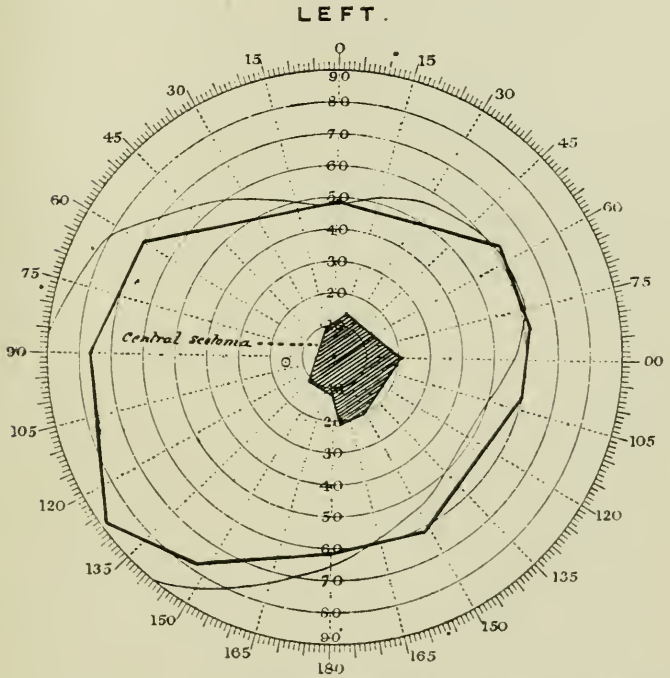
evidence of plugging of arteria centralis retinæ. Condition probably due to retro-bulbar neuritis."

The right optic disc was normal, and there was no hemianopsia.

July 3.—Eyesight improving; counts fingers at one and a half yards. V. = $\frac{4}{60}$.

October 1.—Paresis much improved, but still some inten-

Fig. 1.



G. S., field of vision, taken June 27, 1901. V., left = hand movements with 10 mm. disc.

tion tremor of arm and increased reflexes, with extensor response of right great toe. Vision in left eye now very slightly impaired. V. = $\frac{4}{6}$.

October 25.—Practically no evidence of right hemiparesis remaining, except some exaggeration of right knee-jerk as compared with left. Plantar reflex now normal. Left optic disc still pale.

Case 3. *Left hemiplegia with atrophy of right optic nerve.* —Girl æt. 22 (exhibited before the Clinical Society of London, October 28, 1898).

History.—On March 21, 1898, nine days after giving birth to a child, three eclamptic seizures occurred, attended with loss of consciousness, rigidity, and followed by general convulsions, each fit lasting about two hours. The tongue was not bitten, and there was no incontinence of urine or fæces. The third fit left her hemiplegic on the left side. Several similar fits, apparently without focal commencement, occurred during the following three months; they then ceased. Three months after the first fit she suddenly found that she was almost blind in the right eye. The hemiplegia was at first complete, but partial recovery of power gradually ensued. Her previous health had always been good. The urine was normal, and there was no cardiac affection.

In October, 1898, she was a fresh-coloured, healthy-looking, and well-nourished girl. There was slight loss of power in the left side of the face. The left upper extremity was in the condition of tonic mobile spasm, increased by voluntary movements; the tendon reflexes were exaggerated; the fingers were widely separated, and in constant athetoid movement; the hand was directed to the ulnar side. Tactile sensation was blunted in the hand and forearm, but there was no complete anæsthesia. She could control the spasms to some extent.

The left lower extremity was rigid, the great toe hyperextended, the other toes strongly flexed, so that she walked on the knuckles; the knee-jerk was exaggerated. Ankle-clonus was not elicited owing to spasm of flexor tendons.

The optic discs were examined by Dr. Rayner Batten, who reported the right to be very pale and excavated; vessels small but patent; towards the periphery the fundus was of a good colour. Macula showed fine superficial changes, but no gross changes. Vision was greatly impaired. Right field, tested with large white objects, was decidedly contracted.

She was under observation for three months, during which no changes in her condition occurred.

Remarks.

Case 1 may be said to be of interest firstly on account of the progressive nature of the paralysis, headache, and vomiting from a vascular lesion; and secondly on account of the

sudden and permanent blindness which occurred in the left eye at the same time as the right hemiplegia.

There can be little doubt that the sudden loss of vision, like the sudden hemiplegia, was due to vascular lesion. Whether that lesion was the result of embolism or thrombosis is a matter difficult to decide, and does not appreciably affect the case. The long-standing anæmia suggests thrombosis rather than embolism as the cause.

The microscopical examination of the optic chiasma, nerve, and tracts showed that the whole of the left optic nerve was completely degenerated, and the degeneration could be traced back into the left optic tract, and also across the chiasma into the right optic tract, while the right optic nerve showed no degeneration. There can be no doubt that the degeneration of the optic tract was due and was secondary to the lesion of the left optic nerve, and could not be due to the lesion in the region of the basal ganglia. The question of degeneration of the fibres in the chiasma after an injury to one optic nerve has recently been fully dealt with in a paper by Warrington,* and it is now generally accepted that such a lesion gives rise to ascending degeneration in both optic tracts.

Case 2 is chiefly remarkable on account of the complete recovery from symptoms which seemed at first extremely grave. They suggested a new growth in the neighbourhood of the left internal capsule, and the optic neuritis, slight though it was, seemed in favour of such a view. The boy made no complaint, however, of headache or of vomiting, and the progress of the case negatives the existence of tumour and supports the diagnosis of temporary occlusion of cerebral vessels on the left side. One cannot suppose that the middle cerebral artery was completely blocked, but it is possible that some of its branches—namely, the lenticulo-optic or lenticulo-striate vessels—were occluded, and that there was a similar affection of some branches of the ophthalmic artery supplying the left optic nerve. The occlusion must either have been temporary, or collateral circulation must have been established; otherwise recovery could not have taken place.

Indeed, the results of occlusion of cerebral vessels will largely depend on the abnormal inosculation of vessels which may be present. The greater the abnormality of collateral circulation the better the patient's prospects of recovery will be.

* *Brain*, vol. xxii, 1900, p. 642.

It is difficult to decide whether embolism or thrombosis was the cause of vascular occlusion in this case. The febrile attack with localised pain in the fractured thigh after union had been effected might have been due to periostitis causing thrombosis of periosteal veins. Subsequent detachment of minute emboli which eventually found their way into the cerebral circulation may have accounted for the right-sided hemiparesis. A less probable suggestion is that osteomyelitis in a mild form occurred, and that occlusion of cerebral vessels was produced by fat embolism. It is possible that rheumatic endocarditis caused the febrile attack, and that particles of fibrin became detached from the mitral valve and passed into the vessels of the brain. On the other hand, there was no definite evidence of endocarditis or of rheumatism. The boy was somewhat anæmic; the faint presystolic bruit heard three months after the onset of the paralytic symptoms was probably hæmic in origin, for it soon disappeared. So thrombosis seems the most likely cause of the occlusion of vessels in this case, and such thrombosis of small vessels may have been due to polio-encephalitis.

In Case 3 there was probably occlusion of some branches of the right Sylvian artery. The occurrence of mobile spasm in the left arm suggests that the lenticulo-optic vessel was blocked. The convulsions pointed to similar affection of the cortical branches of the middle cerebral artery.

The ophthalmoscopic appearances were not, in the opinion of Dr. Rayner Batten and other ophthalmic surgeons, due to occlusion of the arteria centralis retinæ, but pointed to the existence of past retro-bulbar neuritis. Probably there was blockage of other branches of the ophthalmic artery supplying the right optic nerve.

The causation of the occlusion of vessels in this case is obscure. Doubtless the puerperal condition was in some way accountable for it. It is said that thrombi sometimes form in the left auricle under these circumstances and may be carried thence to the brain.

On the other hand, the patient's infant was the result of incestuous intercourse between brother and sister. It was stated to have been healthy until three months of age, when it died somewhat suddenly in convulsions. So the possibility that the occlusion of cerebral vessels was due to syphilitic endarteritis presents itself. Against such a view it may be urged that the patient showed no evidence of syphilis, congenital or acquired, but seemed a perfectly healthy young

woman. On the whole we are inclined to believe that embolism was the cause. At the same time, although not anæmic when under observation, she may have been so when the hemiplegia occurred, so thrombosis cannot be excluded.

Other Cases of Similar Nature.

Dr. Hughlings Jackson (*London Hospital Reports*, vol. i, 1864) mentions four cases in which amaurosis on one side was associated with hemiplegia or partial hemiplegia on the other.

Case 4 (p. 374) is that of a man æt. 50 who had complete paralysis and wasting of the right arm, with blindness of the left eye of twelve months' duration. The paralysis and wasting were of gradual onset. The blindness was discovered accidentally. The left optic disc was "quite white, but the vessels were not smaller than they should be." The latter fact, Dr. Jackson thought, "did not countenance the hypothesis that the blindness was due to embolism of the arteria centralis retinæ, although the man had valvular disease."

Case 5.—*Sudden amaurosis; two years later sudden hemiplegia on the left side; mitral disease.*—This was a man æt. 63, the subject of mitral disease, who lost sight in the right eye suddenly two years before an attack of left-sided hemiplegia which lasted a few weeks. The right optic disc was "very white and the arteries small; the veins seemed about the same size as those of the opposite eye." Dr. Jackson's comment on the case is, "Doubtless the amaurosis was due to embolism of the ophthalmic or the arteria centralis retinæ. . . . If the hemiplegia was due to embolism, an artery of the same side of the brain and a branch of the same main trunk was plugged as when the amaurosis was produced. But although there was valvular disease, we must remember that he was from his age, rigid arteries, etc., a likely subject for paralysis from apoplexy or softening."

Case 6 (p. 416) was that of a child (age unstated) who had, simultaneously, defect of sight in the right eye with left-sided hemiplegia. The arteries of the right optic disc were decidedly smaller than those of the left. "The attack came on when the child was scarcely well of measles. There was, when she was brought to hospital, no murmur, but the

mother told us that the doctor said there was something wrong with the heart when the child had measles." Dr. Jackson cites this case as an example of amaurosis on one side and hemiplegia on the other, caused by embolism.

Case 7 (p. 424) is also that of a child, æt. 7, the subject of cardiac disease. Almost suddenly she lost the sight of the left eye and became paralysed on the right side, and died within twenty hours. "The case," Dr. Jackson says, "was clearly one of embolism."

Dr. R. T. Williamson ("Unilateral Retinal Changes in Cerebral Hæmorrhage, Embolism, and Thrombosis," *Brit. Med. Journ.*, vol. i, 98, p. 1515) records thirteen fatal cases of hemiplegia, in eight of which there were unilateral retinal changes on the same side as the brain lesion. The changes were chiefly hæmorrhage into the retina in cases of cerebral hæmorrhage and embolism. He states that in thrombosis of the middle cerebral artery, when the thrombus extends down into the internal carotid, the vessels of the retina on the side of the brain lesion may be markedly diseased and tortuous, while the retinal vessels of the other eye are normal.

In embolism there may be retinal hæmorrhage and slight dilatation of retinal vessels on the side of the lesion.

He does not mention unilateral atrophy of the disc in such cases, but all his cases ended fatally, and, perhaps, before atrophy had time to occur.

The ages of the patients are not mentioned, but it seems probable that they were advanced, or at least that arterial degeneration was present in most of them; whereas in our three cases the ages were 25, 22, and 12 years respectively, and at these periods of life, except where advanced renal disease with arterial degeneration is present, retinal hæmorrhages are not likely to occur.

Reason of Rarity of Occlusion of the Ophthalmic Artery.

There remains the question why blocking of the vessels supplying the optic nerve is of comparatively rare occurrence compared with blocking of the middle or other cerebral arteries. This peculiarity may in part depend upon the position of the branch of the vessel. A vessel given off at right angles from a main trunk—as is the ophthalmic from the internal carotid—is less likely to be blocked by embolism

than one in direct continuation with it, such as the middle cerebral. But it may also in part depend on the anastomosis of the vessel supplying the nerve.

The optic nerve with its sheath receives its blood-supply from a branch of the ophthalmic artery, itself a branch of the internal carotid artery. The nasal branch of the ophthalmic artery passes through the orbit and supplies the inner side of the nose, and there anastomoses with the angular branch of the facial. It is difficult to ascertain in what proportion of cases the anastomosis amounts to inosculation of the two vessels, but after occluding the facial artery by pressure on the side of the nose, pulsation can be felt in the distal portion of the artery, although feebler than formerly, in a large proportion of the cases examined.

Professor MacAlister most kindly replied as follows to an inquiry addressed to him on the subject. He says, "My experience is that failure of distinct union of the vessels is extremely common; certainly in the bodies now in the dissecting room a distinct anastomosis is not present in 10 per cent.

"Such an anastomosis, or the absence of it, might play a very considerable part in the effect produced by an embolism in the ophthalmic artery, for if the anastomosis is free the branches to the sheath of the optic nerve would be supplied through the facial artery, so long as the plug had not passed beyond the point where the branches to the sheath of the optic nerve are given off from the ophthalmic artery.

"Those cases in which sudden blindness supervenes, and no recovery takes place, may be explained either by supposing that the plug has passed so far along the ophthalmic artery as to occlude the vessels to the optic nerve and thus prevent the blood from the facial artery from entering the branch to the optic nerve, or the anastomosis between the ophthalmic artery and the angular branch of the facial is absent, or so small that sufficient blood cannot pass to supply the nerve.

"If the anastomoses between the branches of the facial and ophthalmic are free, that would to a great extent explain the rarity of complete blindness of sudden onset in association with hemiplegia of the opposite side, for the vascular supply of the optic nerve could be carried on through the facial artery, and it would also account for those cases in which rapid recovery takes place. But to explain those cases in which gradual recovery extending over months takes place one must assume that some damage to the nerve has resulted from the temporary block, and that its recovery is only

slowly accomplished, although the vascular supply may be good."

Similar remarks apply to cases of occlusion of the ophthalmic artery or its branches by thrombosis. The effect on the nerve will entirely depend upon the site and extent of the thrombosis and upon the amount of collateral inosculation possible. The suddenness or gradual onset of amaurosis will depend on similar factors.

Our attention has been called by Mr. Armour to a paper by Dr. F. Meyer showing that the variations in the arrangement of the vessels which supply the optic nerve and orbit are very considerable, and that numerous vessels may, under given conditions, enter into the anastomotic supply of the sheath of the nerve. The example given above is probably, therefore, but one of many anatomical explanations which could be given for the complete occlusion of the blood-supply of the sheath of the optic nerve.

In all the cases but one of the series collected the patients either did not come under observation until a considerable time had elapsed since amaurosis had been discovered, or no report of the state of the disc at the onset of the amaurosis is given. In the majority of cases atrophy of the nerve was advanced when the discs were examined. In one case only (Case 2) an opportunity of examining the disc before amaurosis was complained of, was afforded; then distinct evidence of slight retro-bulbar neuritis (engorgement of veins, haziness of margins of the disc) was present three months after the hemiplegic symptoms. But loss of vision was not observed until some weeks later, when atrophy of the nerve was apparent.

In Case 3 Dr. Rayner Batten was of opinion that the atrophy was due to past retro-bulbar neuritis rather than to embolism of the arteria centralis retinæ, and his opinion was shared by several other ophthalmologists; whereas in Case 1 the appearance of the disc was such that it suggested to Mr. Gunn that there was thrombosis of the vessels supplying, or hæmorrhage into, the sheath of the optic nerve.

Conclusions.

1. Atrophy of optic nerve on one side associated with hemiplegia on the other is highly suggestive of occlusion of cerebral vessels on the side of the optic atrophy.

2. Certain features in the cases suggest the possibility of

a neoplasm, and in two of the above cases this idea was for a time entertained owing to the progress and nature of the symptoms—viz. in Case 1 the onset of Jacksonian epilepsy, vomiting, and increasing coma ; whilst in Case 2 the presence of slight optic neuritis on one side led to similar apprehensions.

3. From the appearance of the disc alone in the later stages it would seem probable that the vascular nature of the lesion can be assured. It is of importance, therefore, that the precise nature of these appearances should be ascertained and recorded, in order to aid us in arriving at a diagnosis of such cases in future.

References.

1. Barnes, *Brain*, vol. xxiv, p. 492.
2. Guthrie, *Clin. Soc. Trans.*, vol. xxxii, p. 228.
3. Jackson, *London Hospital Reports*, vol. i, 1864.
4. Meyer, *Morphologisches Jahrbuch von Gegenbaur*, Bd. xii, 1887.
5. Warrington, *Brain*, vol. xxii, p. 642.
6. Williamson, *Brit. Med. Journ.*, 1898, p. 1515.

III.—*Cases illustrating the Clinical Features of an Epidemic occurring at Lincoln in May*, 1902. By W. H. B. Brook, M.D. *Read November* 14, 1902.

IN the early part of May, 1902, an epidemic suddenly occurred in the city of Lincoln, which is worthy of consideration, not only as affording a problem in the tracing of its origin and distribution, but also by reason of its clinical characteristics.

The first case which came to my notice was that of a lady who sent for me on the morning of May 11, complaining of a swollen face and sore throat. This had been noticed early on the morning of the day previous, but so slight was the discomfort that it had not prevented her from travelling to London, returning late that evening. Whilst in London the irritation of the face increased, and on the journey home her face was noticed by a fellow-passenger to become more and more red and swollen.

When seen at 9.30 A.M. she was complaining of intense irritation of the face and upper part of the chest, and also of the throat. The face, especially on the left side, was swollen and erythematous, the erythema extending to the chest, where it was papular and of a rose colour. The tonsils were swollen and the fauces slightly œdematous. There was no albumen in the urine. The temperature was 101·6°.

During the next forty-eight hours the temperature gradually fell to normal and the eruption faded, there being at the same time slight desquamation. On the fourth day the cervical glands were enlarged and tender; this continued for some days, during which time the patient felt weak and pulled down.

For the moment the possibility of having to deal with a case of scarlet fever presented itself to me, but the urticarial character of the eruption, together with its colour and its distribution, which was more marked on the face, was so strongly suggestive of poisoning by food that I asked the patient if she had recently eaten any shell-fish, and was informed that on May 9 she had partaken of some crab, and this appeared at first sight to be a probable explanation of

the illness; but on further inquiry I found that two of the servants in the house were similarly affected with sore throats and a roseolous, papular eruption, occurring in one case chiefly on the abdomen and thighs, in the other being universal in its distribution. In both the tonsils were swollen, intensely congested, of a dark colour, with patches of false membrane, which could, however, easily be removed without leaving a raw surface. These two servants had not eaten any of the crab, so that the cause of the illness had to be sought elsewhere.

One of my partners was at the time attending a severe case of tonsillitis in a neighbouring house, and, meeting him, I ascertained that there were there two patients, both suffering from a similar affection of the throat, in one case with a roseolous eruption, the onset in both having occurred on the previous day, May 10.

During this day and the next I was called to eighteen other cases, all similar in character; all, with one exception, in the same part of the city. My notes of two of these cases, which may be taken as illustrating the general features of the epidemic, are as follows:

Miss M., æt. 32, school teacher. Onset: sore throat and rigors early on May 13. The throat became worse as the day went on. First seen May 14 at my house, complaining of sore throat and of malaise. The tonsils were of a dusky red, swollen, and œdematous, with patches of drab fur which could easily be removed. The tongue was swollen and coated with a similar fur. There was a commencing papular roseolous eruption, with intense irritation on the chest and arms. The temperature was normal.

May 15.—During the next thirty-six hours the eruption became general and lost its papular character, the skin being of a uniform dusky red. The tonsils became more swollen. There was delirium towards evening, the temperature being 102°. There was no albumen in the urine.

May 16.—Eruption fading.

May 17.—Slight branny desquamation, eruption nearly gone, slight enlargement and tenderness of the cervical glands. Temperature normal.

May 18.—Convalescent.

For some days there was pain in the joints, which soon passed off. The patient was able to resume her work on the 26th.

Miss B., æt. 25. First seen on May 13, complaining of sore throat, which had been first noted on the previous day, when she had been sick. The uvula and tonsils were of a dusky red, swollen, and œdematous, but there were no patches of membrane. The tongue was coated. Temperature 101·8°.

The following day there was a slight roseolous, papular eruption on the arms, neck, and chest, which was well out on May 16, when the temperature was 103·8°. There was then tenderness and swelling of the cervical glands.

May 18.—Tenderness of the cervical glands increased; the patient could hardly open her mouth or move her head on account of the pain. At 6 P.M. the temperature reached its highest point, 104·2°, the pulse-rate being 108. The patient looked very ill, was hardly able to swallow, and there was retching and tenderness of the epigastrium.

May 19.—Dr. Gee saw the case in consultation; temperature 102·4°, pulse 102. The eruption was beginning to fade on the arms and chest, where there was a slight branny desquamation. There were patches of false membrane on the tonsils.

During the next week the temperature gradually dropped almost to normal, and during this time there was profuse desquamation. The cervical glands continued tender and enlarged; one suppurated, requiring incision June 12; after that the patient made a slow but steady recovery.

There was never any albumen in the urine. The liver and spleen were never enlarged. Widal's test was negative, nor were any micro-organisms found in the blood. Streptococci were present in cultures of the membrane, but the Klebs-Löffler bacillus was absent. Dr. Klein, however, found a special micro-organism in cultures from the throat, resembling the yeast torula. The patient had had scarlet fever at the age of seven.

On further inquiry we ascertained that all the sick in the practice of my partners and myself had their milk from the same dairy, which led us to think that the cause of the epidemic might be a poison conveyed by milk. This point, however, will be dealt with by others; so far as the clinical aspect of the case is concerned, it is of importance in evidencing the period of incubation.

The matter was brought to the notice of the authorities, and at their request the Local Government Board sent down Dr. Klein and Dr. Darra Mair to investigate the cause of the outbreak.

Cases continued to occur during the week, my partners and myself seeing seventy-five, but we had no fresh cases after May 17—strong evidence that the cause of the outbreak was a temporary one, such as might occur in a milk supply.

In no instance did the disease appear to be communicated by one patient to another; in one house three children who took milk from the particular dairy were affected, whilst an infant of three months, who had milk from another dairy, was unaffected.

This case is of importance because the house was supplied from another dairy, but running short of milk on May 10 they procured some from the suspected dairy. I propose, however, to deal rather with the clinical aspect of the epidemic than with its origin, leaving that to those to whom the investigation was specially entrusted.

The clinical course of the disease was of interest not only on account of its own special features, but also by reason of a certain similarity to scarlet fever, a similarity which gave rise to a report that the epidemic was one of scarlet fever, causing some panic.

The cases under my own care were forty-three in number, and in them the onset of the disease occurred on the following days:

May 9, 6 cases; May 10, 3 cases; May 11, 11 cases; May 12, 4 cases; May 13, 7 cases; May 14, 5 cases; May 15, 3 cases; May 16, 1 case; May 17, 3 cases.

I met with no cases after May 17, and this sudden termination of the epidemic suggests that the poison, whatever its nature, was accidental, and of only temporary duration.

The period of incubation was clearly defined in four cases under my own care, and in one under the care of my partner, Dr. Stanley Green; assuming that, as is most probable, the poison was conveyed by the agency of milk.

1. L. A. M., female æt. 32, the case which I brought first to your notice, habitually had milk from a different supply. On May 11 she took tea with a friend whose milk was supplied from the suspected dairy; in her case the onset occurred early on the 13th, with sore throat and rigors—the throat was worse in the afternoon, very bad all night. A noticeable point is that this patient had taken cream with her tea, and in several other cases the cream seemed to be particularly virulent.

2. S., male under 2½ years old, ordinarily had milk from another source. On the afternoon of the 12th his mother, having attended a parish tea, brought home some of the milk, which had been supplied from the suspected dairy, some of which he drank. He was noticed to be ill and drowsy on the 13th; a roseolous eruption appeared on the 15th.

3. S. F., male æt. 12, only took the milk on one occasion —on May 10, at tea. On May 11 he had rigors and sore throat, and his temperature was 102·2°.

4. B. A. S., female æt. 47, drank freely of the milk on May 11; she felt unwell in the course of the afternoon, and on the 12th complained of sore throat and rigors.

5. P. S., male æt. 20, only took the suspected milk once, on May 14, and was taken ill on the 15th.

These cases fix the period of incubation at from twenty-four to thirty-six hours.

Onset.—Sore throat in every case; rigors, 28 out of 43; vomiting, 6 out of 43; faintness to a greater or less extent in a few cases.

The sore throat.—Great swelling of tonsils, often much more pronounced on one side. In most cases the uvula was swollen; in some it was very œdematous. In some, also, there was swelling and œdema of the tonsils before these showed any change in colour. Colour, dark red—not the bright red of scarlet fever,—and the redness was more marked on the tonsils and posterior wall of the pharynx than on the soft palate, the whole mucous membrane of which appeared swollen and velvety. The tonsils appeared nodular, in most cases the follicles were enlarged, and in many there were patches, some very extensive, of a pseudo-membrane, which was really a thick, drab-coloured fur, like the fur on the tongue, easily removable without leaving a raw surface. In the more severe cases this fur extended over the posterior wall of the pharynx and on to the uvula, the whole of the fauces presenting a horribly sloughy, ragged appearance, but in the majority of cases without any, and in a few very little, destruction of the surface. This fur contained the *Streptococcus pyogenes* and a special micro-organism described by Dr. Klein—resembling the yeast torula, but differing from it in

growing in broth;—never the Klebs-Löffler bacillus, except in one of my partners' cases, where it was found for the first time in the seventh week of her illness. The breath was only offensive in some cases; never so much as in influenza, and the smell characteristic of that disease was never once noticed.

Pain referred to the fauces was in most cases the earliest symptom, and there was difficulty in swallowing, sometimes so marked as to form a serious complication. Great relief was afforded to this pain by the application of a solution of nitrate of silver, and the frequent use of a gargle of weak carbolic acid and borax was most grateful. In all except the more severe cases this swollen and inflamed condition of the tonsils and pharynx subsided rapidly on treatment, although there was a tendency in some cases for it to return if convalescence was checked. In only one of my cases was the tonsillitis of a suppurative character, although instances of quinsy were met with by others.

Temperature.—Never very high when first seen within a few hours of the onset, ranging between normal and 102·4°. In one case of a child æt. 7, the temperature, which was 101° the first day, rose to 102·6° on the second day; but in most of the other cases the temperature fell on the second day with the appearance of the eruption, and on the third day was in most cases below 100°, and in many normal, even with a very profuse eruption. This course of the temperature only obtained in uncomplicated cases; in some it again rose on the fourth day, concurrently with adenitis of the anterior chain of cervical glands, or with symptoms of gastric irritation. The general tendency was, however, for the temperature to fall on the appearance of the eruption.

The pulse was febrile in character, but was never excessively frequent; in most cases its rate fell below the ordinary ratio of pulse to temperature (+ 10 beats for each degree above the normal). It was rarely, except in little children, above 102, and even in cases where the temperature reached 104·2° was only 112, an important point in the differential diagnosis between this disease and scarlet fever, in which the pulse-rate is increased out of all proportion to the temperature.

The tongue was flabby and soft, coated with a thick drab fur, which could be easily scraped off; not dry except in the worst cases, tending gradually to clear at the edges. The tongue more resembled that of influenza; in none of my cases was there a "strawberry" or "raspberry" tongue.

The eruption was met with in 27 cases out of 43. It appeared in twenty-four to thirty-six hours after the onset, mostly first noticed on the neck and chest, occasionally on the face; in some cases first on the abdomen and thighs, in some on the forearms and feet. It was never scarlet, nor was it a fine, punctate eruption, but it resembled that of Rötheln—crimson papules the size of a large pin's head, which projected sufficiently to be felt, the intervening surface being covered with an erythema of the same colour, but of a paler shade. In some the whole eruption became confluent, the patient looking as if he had been dipped from head to foot in a crimson dye. In many cases the erythema was accompanied with intense irritation; in two it was of a distinctly erysipelatous character, and in one it was strongly suggestive of urticaria, and in some there was œdema of the fingers and hands. The eruption may well be compared with that met with in cases of shell-fish poisoning, and I have also seen it in two cases of puerperal fever.

The duration of the eruption varied between a few hours and a week; in the latter cases, as it disappeared it left a brown stain, showing that there had been some extravasation into the papillæ. In one patient there had been no eruption, but an intense hyperæsthesia of the arms and chest.

The disappearance of the eruption was *accompanied with desquamation*, the character of which varied with the degree of fever and the intensity of the rash. It began first about the neck and shoulders, then in the axillæ and groins. In most cases it was merely furfuraceous, but in some there was extensive shedding of large flakes. It began very early in the course of the disease, as soon as the rash began to fade, and several days before it finally disappeared—an important point of distinction between this epidemic and scarlet fever, in which desquamation does not commence, as a rule, until some days after the rash has faded.

In none of my cases was there desquamation without a previous rash. The nose was not affected, nor was there, except in one case, suffusion of the conjunctivæ.

A symptom so frequently met with (in 25 out of 43 cases) as to be almost a regular part of the disease was inflammation with considerable swelling and exquisite tenderness of the anterior chain of cervical glands, extending in some to the clavicular, but not to the submental glands. This was more marked on that side on which the tonsil had been more affected. This adenitis was generally present

to a slight degree on the third day, but there was one circumstance which greatly aggravated it, namely, a too early return to meat diet; and this was so even when the patient had apparently quite recovered. The occurrence of *marked* cervical adenitis, which was never before the fourth day, was accompanied by a rise in temperature to 102°—104°, and if considerable by a return of the tonsillitis and by tenderness and stiffness of the neck, so that in the most severe cases the patient was in a most pitiable condition, unable to move the head, and only swallowing with great difficulty. As is so often the case, a thickened food was the most easily swallowed. This difficulty in swallowing in extreme cases was marked by great terror, shown both in the expression of the face and also by the patient's fear of being left alone.

Stiffness of the muscles of the neck, apart from the cervical adenitis, was not infrequently met with, but there was never any retraction of the neck. The bowels were constipated; there were anorexia and thirst, with a tendency in the more severe cases to delirium at night. In no case was there any affection of the lungs or heart, except a tracheal cough in two children and some bronchitis in two old people, who were, however, always subject to a winter cough. The urine did not contain albumen, except in one case in which hæmaturia appeared at the end of the fourth week, and this also was the case in a patient under my partner's care.

The *recovery* in uncomplicated cases was rapid, the patient feeling well by the end of a week, although somewhat weak and pulled down; but there was a certain tendency to relapse, and in those cases in which complications occurred convalescence was very tedious, and often accompanied by several relapses, so that it extended over a period of several weeks.

Complications of the disease.—The most frequent complication was gastric irritability and sickness, passing in extreme cases to gastritis, and in one that was brought to my notice to fatal peritonitis. This gastric irritability was avoided by keeping the patient to liquid soft food, and by the avoidance of alcohol except in very small quantities, well diluted; if alcohol was needed in such cases, champagne appeared to be the best form.

Stiffness of the muscles and joints was noticed in many instances, especially as affecting the knee and fingers; it appeared due rather to a thickening of the structure round the joint than to any changes in the joint itself. But in certain cases this complication, occurring during convalescence, took

the form of rheumatic fever, the joints being swollen and tender, the patient lying quite helpless, unable to move hand or foot, suffering after the slightest movement; with this was pain in the muscles, notably at the insertion of the deltoid. This was accompanied by redness in patches on the elbow, knee, ball of great toe, and dorsum of foot, and by a rise of temperature to 103°. This condition did not absolutely resemble rheumatic fever; there was no sour smell and no profuse sweating, nor was there ever any sign of endocarditis, but in one case (that in which the Klebs-Löffler bacillus was found) there was great infrequency of the pulse (forty-eight beats to the minute) and a tendency to faint if the arms were raised.

Septic phlebitis was met with in one case, which terminated fatally on the twentieth day, with symptoms of pyæmia, death being ushered in with hemiplegia, coma, and convulsions. Apart from the presence of the specific micro-organism which is described by Dr. Klein, certain clinical details were noted, viz. only twice was albumen found in the urine, and then late in the disease and in association with hæmaturia. In one case in which the blood was examined Widal's reaction was negative; no micro-organisms were found in the blood, which showed an increase both of coloured and colourless corpuscles, 1 cubic mm. containing 6,925,000 coloured corpuscles, the leucocytes being present in the proportion of 1 in 400.

The method of treatment that was found to give the best results was that which was adopted from the general nature of the disease. At the first onset of the illness the patient was kept warm in bed, in some cases rather unwillingly; he was fed on liquid food, with plenty of water, to flush the poison out of the system; and with the same object calomel, followed by a saline purge, was given when first seen.

As a general rule a simple saline mixture, with salicylate of soda, was given so long as there was fever, followed during convalescence by quinine and strychnine in combination with dilute hydrochloric acid, or in the Mist. Chlori, which had a good effect in cleansing the tongue.

The local treatment, though simple, was of considerable importance, judging from the benefit that the patients seemed to derive from it. The pain and swelling of the tonsils was best lessened by the application daily for the first few days of a solution of nitrate of silver of the strength of one drachm to the ounce; and the general discomfort about the fauces was relieved by the frequent gargling with a solution of glycerine of carbolic acid and glycerine of borax.

The gastric irritability was best treated by bicarbonate of soda and dilute hydrocyanic acid, with counter-irritants to the epigastrium.

For the enlarged and tender glands in the neck, belladonna fomentations afforded most relief early in the case, followed as the temperature subsided by the application of glycerine and belladonna on cotton wool, and later, as the tenderness subsided, by gentle friction with a liniment composed of equal parts of Lin. Belladonnæ and Lin. Camphoræ.

Bearing in mind the excellent effect of Llangammarch water on enlarged strumous glands, I gave it steadily in several instances, and its administration appeared to assist in the diminution of the glands.

The pain and stiffness of the joints was best treated with salicylate of sodium, and by occasional doses of antipyrine.

Fever was met by tepid sponging and antipyrine, and where it was excessive it was at once reduced on the administration of the antistreptococcus serum.

As has already been mentioned, a return to a meat diet had to be watched most carefully, and the same may be said of the administration of stimulants, which, if given too early, were apt to be followed by increased pain in the throat and glands, but later in the disease they were required after the temperature had fallen, but not as a rule before the rash had completely faded. Brandy was the most useful form of stimulant, but if there was gastric irritability, then champagne had a good effect.

Mortality.—None of the patients under my own care died, but one under my partner's care died with septic phlebitis, hemiplegia, and coma.

Points of distinction between the symptoms of scarlatina and the Lincoln epidemic:

Symptoms of scarlatina.	Symptoms met with in the Lincoln epidemic.
Onset. — Throat symptoms rarely severe at first.	Throat symptoms most marked from the very first.
Fauces bright red; tonsils not generally very swollen at an early stage.	Fauces dark red; great swelling of tonsils, with patches of false membrane; œdema of the uvula.
Generally vomiting.	Vomiting comparatively rare.
Eruption.—Scarlet; punctate; appearing first on chest; rarely well marked on face.	Crimson; papular; well marked on face; sometimes first seen on forearms.
Tongue.—"Strawberry;" later "raspberry."	Influenzal tongue; never "strawberry," but soft, flabby, and large.
Temperature.—Falls gradually as the eruption fades.	Falls on the appearance of the eruption.

Symptoms of scarlatina.	Symptoms met with in the Lincoln epidemic.
Pulse.—Frequency increased out of proportion to the fever.	Frequency not so much increased as would be expected from the degree of fever.
Desquamation.—Does not begin till after the eruption has faded, often not for some days after.	Desquamation commenced whilst the eruption was still out.
Urine.—Often albuminous.	Albuminuria only twice in seventy-five cases, and then in the fifth week, with hæmaturia.
Age.—Most common in early life.	Occurred in many old people.
Incidence.—Second attack rare; return cases frequent.	Occurred in many patients who had had scarlet fever; no return cases.
Rheumatic symptoms comparatively rare.	Comparatively frequent.

These points of distinction will, I think, prove that the Lincoln epidemic was not only quite distinct from scarlet fever, but was, in fact, a special disease—a conclusion which, arrived at here on the grounds of the clinical evidence, will, I believe, be borne out by the results obtained by those who have investigated it from the standpoint of the bacteriologist.

In conclusion I have to acknowledge my indebtedness to my partners, Mr. Charles Brook (to whom was due the suggestion, in the first instance, that we were dealing with a specific malady) and Dr. Stanley Green, for the opportunity of examining and comparing cases under their care; to Dr. Klein and Dr. Darra Mair for many valuable hints as to the method of investigation; and lastly, to you, gentlemen, for so kindly allowing me to bring this paper before you this evening.

IV.—*Cases of Gastrotomy for recent Gastric Ulcer.*

By C. Mansell Moullin. *Read November* 14, 1902.

THE first case was that of a woman æt. 30, who was admitted into the London Hospital for hæmatemesis on May 13, 1902. The history was to the effect that she had been subject to vomiting occasionally for the last twelve years, and had suffered from dyspepsia, but not to any severe degree. Seven weeks previously she had been confined. She was not suckling.

Seven days before admission she had fainted without any apparent reason. The following day she fainted again, but on this occasion she vomited half a pint of blood. She was put to bed and kept on milk diet. Two days later she vomited again, bringing up a pint of blood; and this happened again, about the same quantity being brought up, the evening before she came in. On admission it was noted that she was exceedingly anæmic, but not wasted. Pulse 120; temperature 100·8°. There was no abdominal tenderness or pain. The abdomen was soft and flaccid, the liver not enlarged, and the heart and lungs were clear.

As she had vomited a considerable quantity of blood on three occasions within a week, not counting the first occasion on which she had fainted, and was very much blanched, it was decided, after a consultation with Dr. F. J. Smith, to operate without further delay. When the abdomen was opened nothing abnormal could be seen on the peritoneal surface of the stomach. An incision was then made through the anterior wall and the interior carefully examined bit by bit, partly by everting it, partly by introducing a vaginal speculum, as I have described on a previous occasion. A small abrasion was found on the anterior wall, not penetrating the whole thickness of the mucous coat. Blood was pouring from it freely. A silk suture was passed under it, backwards and forwards, and then partially around it, so that when the thread was tightened the whole of the abraded surface was tightly drawn together. The anterior incision and the abdominal wound were then closed in the ordinary way.

The subsequent course of the case requires little comment.

Strychnia and caffeine were given freely hypodermically. Hot water was given by the mouth as soon as she had come round from the anæsthetic, and small quantities of milk at the end of twelve hours. The day after the operation she was sick three times, and the following day once. Nutrient enemata were given for forty-eight hours, at the end of which time the bowels were opened by means of calomel and castor oil. The patient was put on full milk diet on the third day, and left the hospital at the end of three and a half weeks, having been for some time on full diet.

The second case was that of a woman æt. 27, who was also admitted for hæmatemesis. The first attack had been two years before, when she was an in-patient for some time. Since then she has suffered continually from dyspepsia. On the day before admission she was sick twice, on the second occasion bringing up (so she stated) two quarts of blood. The next day, while in hospital, she was sick again, though she had had nothing by the mouth, and on this occasion she did bring up approximately a pint of blood. In addition a good deal was passed by the bowel. The pulse was regular, 112; the lips and face were pale, and the patient was a good deal collapsed. After consultation the patient was transferred for operation the next day.

On inspection of the serous surface of the stomach a spot was found on the posterior surface near the cardiac end of the lesser curvature, where the peritoneum was thickened and opaque, and the mucous coat could not be easily separated from the muscular one by pinching the latter up with a fold. On opening the stomach and inspecting the interior through a vaginal speculum, a chronic ulcer about the size of a three-penny piece was found at the corresponding spot. The ulcer itself was punched out, and the base was formed of the muscular coat. The edges were indurated, all the coats of the stomach being matted together, but not much swollen. The base of the ulcer was a greyish yellow in colour, and no bleeding point was seen. The ulcer was grasped from the mucous surface with a pair of forceps, drawn into the interior of the stomach, so that it formed the apex of a cone, and then a stout silk ligature tied as tightly as possible around the base of the cone, so as to strangulate the invaginated portion. The anterior wound of the stomach was then sewn up, and several sutures passed through the sero-muscular coat opposite the base of the ligatured cone, as an additional precaution.

An injection of about a pint of beef tea, brandy, and coffee was given by the rectum, and strychnia and caffeine hypodermically. Small quantities of iced champagne, and milk and water, were given by the mouth the same evening, and nutrient injections were ordered at frequent intervals. The second day the patient vomited six times, and two grains of calomel were given and repeated. The bowels were opened at the end of forty-eight hours by enema, and as there was no more vomiting the patient was given jelly and custard on the third day. On the sixth day she was put on reduced iron, and by the tenth day she was taking fish and eggs; but it was some time before she got over the anæmia. Ultimately she left the hospital, able to take ordinary diet without inconvenience or pain.

The third case was that of a woman æt. 24, the immediate cause for operation being again hæmatemesis. There was a history of an attack two years previously, and of indigestion ever since. She was admitted on July 11, 1902. Six days before that date, and again the day before, she had vomited half a pint of blood. On the 12th she vomited again about the same amount; and there was continuous melæna. The patient became very anæmic, yawning, and restless, the pulse-rate running up to 140; and on the 14th it was decided to operate. The lesion found was almost identical in shape and position with that in the last case, but on attempting to pass a ligature around the inverted ulcer the mucous membrane was so softened that the ligature cut through, and the walls were so thickened that, in spite of two attempts, it would not hold. Accordingly sutures were passed from the mucous surface through and through the base of the ulcer, and the contiguous ends of the sutures were tied together so as to effect approximately the same result. Additional sutures were passed through the sero-muscular coat as before, and the wounds in the anterior wall of the stomach and the wall of the abdomen were closed in the usual way.

The patient was very much collapsed. She had lost a very large amount of blood, chiefly by the bowel, and owing to the ligature not holding the operation had taken nearly an hour. Mr. Lett, the surgical registrar, transfused two pints of fluid into the basilic vein during the operation, and a pint and a half more was poured into the peritoneal cavity; but, in spite of this, and a very large enema (more than a pint) of coffee, beef tea, and brandy, my house surgeon, Mr. Moore

Ede, to whom I am indebted for these notes, found her so collapsed that evening that he had to transfuse her to the extent of three pints more. After this she rallied, and for the first four days everything went on well; the vomiting ceased and the bowels were opened freely. On the morning of the fourth day peritonitis set in. The same afternoon she was seized with a sudden pain in the right iliac fossa. The next day vomiting set in again. The abdomen remained soft and flaccid and moved with respiration, and the bowels continued to act, but the patient grew weaker and weaker; the lower part of the abdomen became tender on pressure, and it was evident that peritonitis had set in. Reopening of the abdomen was contemplated, but the pulse was too weak, and the patient died on the eighth day after the operation. At the *post-mortem* there was general peritonitis, due to the escape of the contents of the stomach through the anterior wound, which showed no sign of healing, the stitches having cut their way out in several places. There was no leakage from the posterior wound.

The fourth case was that of a woman æt. 25, who was admitted under the care of my colleague, Dr. F. J. Smith, for hæmatemesis on July 24, 1902. She had had three previous attacks, at intervals of about six months, and on each occasion had been kept in bed for weeks and fed on nutrient enemata. The present attack had commenced fourteen days before with pain and vomiting. Blood had been only vomited once, but there was a great deal of melæna. After consultation the abdomen was opened. There was a thickened patch on the peritoneal surface near the pylorus, and on opening the stomach two recent acute ulcers were found close together, not extending into the submucous layer. In addition there were several blackish spots, indicating the sites of old hæmorrhages. The ulcers were secured by silk sutures passed under their bases and around them from the mucous surface, and the wounds closed in the usual way. After the operation the patient vomited four times. Milk was given by the mouth in small quantities the same evening; nutrient enemata were kept up for two days. On the sixth day custard and jelly were given, and a few days later, bread and butter and pounded fish. The patient left the hospital three and a half weeks after the operation, having had no further pain or vomiting.

The fifth case was that of a woman æt. 23, who was admitted under the care of my colleague, Dr. F. J. Smith, on

April 7, 1902. For some years she had suffered from indigestion. Four years previously she had been treated for gastric ulcer. One week before admission she had been seized with sudden pain in the left epigastric region. There was no vomiting, but the pain had continued, and was especially severe after eating or drinking. The left rectus was distinctly more rigid than the right. Dr. F. J. Smith diagnosed a gastric ulcer, with an adhesion to the anterior abdominal wall. As the patient did not improve in the course of a fortnight the abdomen was opened, and the diagnosis exactly confirmed. The adhesion was divided; the ulcer inverted and sutured. After the operation the patient vomited three times. On the third day she was put on full milk diet. At the end of a week she was given pounded fish; and at the end of three weeks she was discharged free from pain, having been for some days on full diet.

The sixth case was that of a girl æt. 20 years, who was admitted under the care of Dr. Sansom in April, 1902, complaining of vomiting, which had persisted every day, and often several times a day, for upwards of six months. There was a history of gastric ulcer four years before, and of repeated attacks of hæmatemesis since. On admission it was noted that she was not wasted, and that there were no physical signs of disease. There was tenderness in the left epigastrium, and, after taking food, severe pain until she vomited. This pain and vomiting occurred regularly until all food was stopped by the mouth. After ten days with nothing but water, milk was tried, with the result that the pain immediately began again. As nothing did any good, exploration was advised. The only lesion found in the stomach was a small abrasion on the anterior wall. This was sutured and the wounds closed in the usual way. The patient was given milk as soon as the vomiting caused by the anæsthetic was over; by the fourth day she was on full milk diet; at the end of a fortnight she was taking fish; and she went out three weeks and a half after the operation, able to digest full diet without pain, though she was still sick occasionally. Since then I have heard that she has relapsed, and that the old pain and vomiting have returned.

As regards this case, I am still uncertain as to whether it was a case of recurrent ulceration which should have been treated by gastro-jejunostomy, or whether it was simply functional. The patient was singularly well nourished for one who suffered from so much pain and vomiting. There was no

cicatrix, such as I expected to find. There was no hæmatemesis while in hospital, in spite of frequent vomiting, though there was a history of its having occurred repeatedly before she came in; and her complaint, whatever it was, was associated with an extreme degree of intestinal inertia. After the operation, not only were the nutrient enemata and two large injections containing turpentine retained without discomfort, but it took sixteen grains of calomel, and more than two ounces of castor oil, before an action of any kind could be obtained. The cessation of the symptoms during convalescence, and their recurrence within a few months of her discharge, are consistent with either hypothesis.

The seventh case is of especial interest in that I could not find the ulcer in the stomach. The patient was a man æt. 35, who was admitted under the care of my colleague, Dr. F. J. Smith, for hæmatemesis and repeated melæna. The interior of the stomach was thoroughly examined, but no bleeding point or ulcer was discovered. The patient recovered and went out, and remained perfectly well for nearly a twelvemonth. Then suddenly, about a fortnight ago, he was seized with faintness, all the signs of severe hæmorrhage, and profuse melæna. There was and had been no vomiting of any kind; and from this and his previous symptoms I think there can be no doubt that the actual source of the bleeding is in the duodenum and not in the stomach.

An eighth case, that of a man æt. 53, who has suffered from hæmatemesis and melæna off and on for upwards of eight years, and upon whom I performed gastro-enterostomy six weeks ago, has left the hospital, apparently perfectly well; but of course the time is too short for the case to be of any value.

In addition to these eight cases I have been asked, chiefly through the kindness of my colleagues, Dr. Sansom and Dr. F. J. Smith, to see in consultation during the last three years, eleven others. In five of these I did not operate. Three of them were cases of hæmatemesis, and were practically dying, beyond even transfusion; and they all did die in the course of twenty-four hours. The fourth I regret exceedingly. The patient, a young woman, was under my care in the hospital for a month. During that time she did not exhibit a single one of the ordinary symptoms. She was only sick once, and never brought up any blood. After the first day or two she did not suffer from pain, and at the end of a month she went out apparently perfectly well, on ordi-

nary diet. Six months later Mr. Cuthbert Dixon, of Mare Street, Hackney, wrote to me saying that she had been admitted into another hospital for acute perforation. Whether this was the same ulcer as that from which she suffered when she was in hospital under my care is an open question. My impression, for reasons that I will give later, is that it was not; that it was another, which had developed subsequently; but I cannot help thinking that if I had operated on the first occasion, and performed gastro-jejunostomy, the perforation would not have occurred. The fifth case refused operation, and she is, or rather was a month ago, still alive, if such an existence can be called life; for Mr. Charles Hirsch, of Woolwich, who wrote to me about her, said that she was reduced to the state of a skeleton, completely bedridden, unable to lift hand or foot, and far too ill for any surgical treatment. Whether she is still alive or not I cannot say.

The remaining six were operated upon, one because of pain and vomiting, the other five (one of whom died) for hæmatemesis. The notes of these have been already published.

Putting aside, therefore, all operations for the complications and sequelæ of gastric ulcer, such as perforation, pyloric stenosis, hour-glass contraction, perigastric abscess, adhesions, and such like, of the nineteen cases in which I have been consulted with a view to operation, the five which were not operated upon are dead, or as good as dead; and only two of the other fourteen. In eleven of these fourteen the immediate cause for operation was bleeding, and six of these were so far gone from loss of blood that transfusion had to be performed upon the table or immediately after. Of the two that died, in one (already published) the kidneys were in a state of advanced interstitial nephritis, and there was an open artery on the floor of the ulcer; in the other, as already mentioned, the loss of blood had been so great that the patient had not sufficient recuperative power left to effect repair of the wound. For myself I am convinced that if the operations had only been performed at an earlier period, before the patients had lost such an amount of blood, the result would in all human probability have been different.

I may at once disarm criticism by saying that I have not the least intention of advocating operation as a general mode of treatment for ulcer of the stomach, or of removing such cases from medical wards. Ulcer of the stomach is, and always will be, more medical than surgical. But having said this much, I cannot help thinking and saying that if a good

many of these cases were treated by operation, not only would many more lives be saved, but a vast amount of suffering would be avoided.

Such a statement should, I suppose, be supported by statistics. With this object in my mind I requested Dr. C. V. Bulstrode kindly to collect and analyse for me the statistics of all the cases of gastric ulcer admitted into the London Hospital from 1897 up to the present time. They were just 500 in number, 98 men and 402 women. Out of this number no less than 48 (nearly 10 per cent.) died from perforation and peritonitis; 13 (2½ per cent.) from hæmatemesis; and 28 (5½ per cent.) from other causes. The total number of deaths was 89, or approximately 18 per cent. Now these statistics comprise the whole of the cases of gastric ulcer admitted into the hospital, mild and severe alike. Therefore they require a certain amount of examination. No one, so far as I am aware, proposes, or ever has proposed, operating upon all cases of gastric ulcer indiscriminately. So far as the majority is concerned, no question as to the propriety of operation could ever arise. All these cases, therefore, must be ruled out. They do not enter into the argument. The only cases that can be considered are those in which the condition is so grave that the possibility of an operation has to be taken into consideration; and the only comparison that can be made with any pretence at fairness is between those cases in which an operation is thought to be advisable and is performed, and those in which it is thought to be advisable and is not performed. Taking the whole of the cases, grave and mild alike, together, the mortality is 18 per cent. If all the slighter cases, those in which no one would dream of operating, are eliminated, and only the gravest of the grave are reckoned, the mortality would undoubtedly be far higher, how much higher I should not like to say; but I have no hesitation in saying that it would be far higher than that which would follow if all these grave cases, and not merely the selected ones, as I am advocating, were operated upon at once.

It is not, however, only the immediate death-rate that requires to be taken into consideration. I maintain that, if these cases were operated upon, not only would the mortality be materially less, but the patients who survive would be saved from an enormous amount of suffering, both bodily and mental. Excision of a gastric ulcer has at least the merit of being final. If the patient recovers he recovers completely.

Another ulcer may form if the condition which gave rise to the first continue the same, but relapse is not possible. I thought it would be of interest, in connection with this question, to try to find what proportion of cases relapsed under the present method of treatment, and I asked Dr. Bulstrode to try and make this out for me. This manifestly could not be done; it involves an inquiry into the future. But he very kindly took the trouble to go into the history of these patients, and he found that out of the 500 no less than 214, or 42 per cent., had suffered in this way previously. In 116 there had only been one previous attack; in 41 there had been two; in 15, three; and in 39, four or more. In other words, gastric ulcer recurs, or relapses, in at least two fifths of the cases that are apparently cured. As the majority of these patients are living still, of course the actual number, if it could be ascertained, would work out a great deal higher.

These figures, collected for me by Dr. Bulstrode, do not include what may be termed the late sequelæ of gastric ulcer, such as cases of pyloric stenosis, dilatation of the stomach, and other troubles due to the presence of cicatrices; nor do they include the cases, estimated by Hauser at 6 per cent., in which carcinoma develops at the site of an old chronic ulcer. If allowance is made for these—for the 18 per cent. of deaths and the 42 per cent. of relapses,—it must be admitted that the proportion of cases of severe gastric ulcer which are cured, in the full sense of the term, is not a very high one, not nearly so high as is usually imagined; and that there is quite sufficient justification for operation, at any rate in selected cases.

With regard to the method of operating in these cases, I need not enter into details, as I have already published them. The mode of treatment of the ulcer must depend upon its size and extent when it is exposed. In two cases I have been satisfied with strangulating it by means of silk sutures passed underneath and around it from the mucous surface. In three I have excised the ulcer without touching the muscular coat, and sutured the cut edges of the mucous membrane together again. In one I excised a portion of the whole thickness of the wall of the stomach. In two others, both of which were successful, I ligatured the whole thickness of the wall of the stomach, strangulating the portion that contained the ulcer by tying a silk ligature around it from the mucous surface as tightly as I could, and then placing a few Lembert's sutures, as a precaution, upon the serous surface. And in one case,

as already described, in which this was not possible, I carried out the same idea by transfixing the stomach wall and tying the ends of the ligatures together. This patient died, but this wound had not leaked. Gastro-enterostomy, it seems to me, should be reserved for cases in which the ulcer cannot be dealt with directly, either from its size or its position; for those in which the source of hæmorrhage cannot be found; and for those in which the ulceration is recurrent—a class of case of which I wish to speak later. It is not a good operation for hæmorrhage when the source of the bleeding can be found. In two cases, at least, the bleeding has continued and has proved fatal, in spite of gastro-enterostomy having been performed. If the bleeding point can be found it should be dealt with in the ordinary way.

The indications for operation depend upon whether the ulcer is acute or chronic. In the case of the former there are only two—threatened perforation and hæmorrhage. When perforation has already taken place the operation is performed because of the perforation, and not because of the ulcer. An ulcer should be made secure before it has perforated.

Our knowledge of the premonitory signs of perforation is very small. There may be none. The wall of the stomach is, for the most part at any rate, entirely devoid of ordinary sensibility. It may be cut, or pinched, or burnt without the patient being aware of it; and therefore, of course, it may as readily ulcerate through without giving any warning sign. The severity of the pain after food, and the time when the pain begins, are of little or no value. The former is very difficult to estimate, and may be as great in simple dyspepsia; the latter depends upon the part of the stomach involved, the energy of its movements, and the rapidity with which the neighbouring sensitive portion of the parietal peritoneum becomes irritated. It will occur, for example, earlier when the cardiac end is involved, owing to the proximity of the diaphragm and the readiness with which the sensitive peritoneum upon its under surface becomes irritated, than when the ulcer is at the pyloric end. But I am disposed to attach considerable importance to local tenderness on pressure, especially when it is associated with cutaneous hyperæsthesia in Head's epigastric triangle. This is an indication that the irritation spreading from the sore is sufficient to cause persistent tenderness of the neighbouring portion of the parietal peritoneum, a tenderness which is reflected down the

cutaneous branches of the corresponding spinal nerves. When this is present and well marked, and especially when it persists in spite of treatment, it may be taken that the ulcer is in a state of irritation, and that it is not only not healing, but is spreading, and therefore is likely to end in perforation. But the absence of local tenderness means nothing.

Hæmorrhage, in the case of an acute gastric ulcer, is described as either capillary, venous, or arterial; and it is generally stated that there is no means of telling one from the other. From the cases, however, in which I have operated myself, I am strongly disposed to believe that bleeding, when at any rate it is severe, is nearly always, if not always, arterial in origin. During life the ordinary acute ulcer of the stomach usually takes the appearance of a linear or triradiate fissure, out of which the blood trickles in a steady, continuous stream. The classic funnel-shaped description is only found *post mortem* when all the congestion has subsided. The sides of a recent ulcer are so swollen from inflammation and œdema that during life they meet over it and close it in. Hence it is that the blood that is poured out in its depths can only escape slowly, and shows no pulsation. *Post mortem*, when all the congestion and œdema have gone, the arteries, especially in a patient who has died from loss of blood, contract to such an extent that they appear to be quite insignificant in point of size, and a lateral opening, such as is usually present, is almost certain to be overlooked. Unless the blood-vessels of the stomach are injected, or some special examination is made, the bleeding point is scarcely ever found in an acute ulcer, and the loss of blood is almost certain to be laid to the credit of the capillaries.

A first hæmorrhage in the case of an acute ulcer of the stomach is very rarely fatal. If, however, it is at all serious, and is followed by a second attack within a few days, in spite of absolute rest, I believe the bleeding point should be exposed and dealt with on ordinary surgical principles. Some vessel is bleeding. The chances are that it is an artery, and that there is an opening in one side of it, which notoriously does not close easily. The bleeding has stopped once. The clot that plugged the opening has been digested away or is gone. It is not safe to run the risk of a third serious hæmorrhage. Provided the conditions are suitable and adequate assistance is at hand, the danger of operation is the lesser of the two. Laparotomy at the present day, under these circumstances, is perfectly safe. Opening the stomach, if

done with proper precautions, does not add materially to the risk. There remains the difficulty of finding the bleeding point, which is much easier during life than after death, and of dealing with it. It is only in the case of chronic ulcers of large size and numerous adhesions, cases that have been allowed to drift, that this difficulty is material.

The indications in chronic ulcer of the stomach are entirely different. The mere fact of its being chronic is proof that all previous treatment has failed, and if it has failed when the ulcer was small and recent it is not likely to succeed when it is old and large. Chronic ulcer of the stomach should, I believe, in all cases be treated surgically, provided one good attempt at cure has been made and has not succeeded. The longer it lasts the more the local conditions which attend it, such as the induration about its base and its adhesions to surrounding organs, prevent its healing. It gives rise, if it is left, to an endless series of complications, many of which are fatal, all of which cause great misery, and all of which could be avoided by timely operation. It leads to cancer. Often, at last, it gets so bad that no operation is possible. And of itself it is very fatal. Some time ago Dr. Wall collected for me the statistics of the cases of gastric ulcer admitted into the London Hospital during five years. Only those suffering from hæmatemesis were counted, and, as far as possible, all other causes than ulcer of the stomach were excluded. Of those over thirty years of age no less than 6 per cent. of the women and 12½ per cent. of the men died from this cause alone. If to this is added the number of deaths from perforation, exhaustion, subphrenic and perigastric abscesses, and the numerous other complications that occur in connection with chronic ulcer, it must be admitted that the total death-rate is a very serious one, far higher than would be the case after timely operation.

Finally, there is another class of case in which I consider operation to be advisable—a class which hitherto has been confused with that in which there is a chronic ulcer of the stomach. Its characteristic is the constant recurrence of ulceration. There appears to be some morbid state of the stomach, or of the constitution generally, as a result of which ulceration of the stomach occurs time after time. That patient whom I have already mentioned, who left the hospital and died six months afterwards from acute perforation, I believe to be an instance. That other patient in whom, when I opened the stomach, I found the usual recent bleeding

fissure and several old hæmorrhagic spots in the mucous membrane near, certainly was one. She had had four separate attacks of hæmatemesis separated from each other by intervals of months of perfect health. It is this that distinguishes these cases from those of chronic ulcer. A patient with chronic ulcer is never really well; these patients are. They are able to do as they like in the intervals. I have operated upon several such, and in all, instead of the chronic ulcer which I had expected to find, I have found a recent acute one, bleeding freely. These I have ventured to put by themselves as cases of recurrent ulceration of the stomach. The name is not a good one, as it describes what is really a symptom, and says nothing of the condition which underlies and causes that symptom. What that condition may be I do not know. It is not simple hyperchlorhydria, for that may not be present at all; but whatever it is, when it persists and cannot be stopped by ordinary measures, and is perpetually placing life in danger by causing ulceration, it should be treated by gastro-enterostomy, or some other device by which the stomach can be given complete rest until it has had time to recover.

V.—*Ruptured Crucial Ligaments and their Repair by Operation.* By A. W. Mayo Robson, F.R.C.S. *Read November* 28, 1902.

AT first sight it would appear that an accident capable of rupturing the crucial ligaments of the knee would lead to complete disorganisation of the joint; but the case that I am about to relate demonstrates not only that the crucial ligaments may be ruptured without serious damage to the joint as a whole, but that their repair by operation is both feasible and hopeful as to its ultimate result.

J. B., æt. 41, a miner, residing at Featherstone, was admitted to the General Infirmary at Leeds on November 4, 1895, on account of lameness from weakness and instability of the right knee, resulting from an accident when at work in a coal-pit thirty-six weeks before, when he was almost buried by a fall of earth, and sustained, besides the injury in question, fracture of the left leg and of three ribs on the left side, as well as internal injuries. After being in a neighbouring hospital for six weeks he was made an out-patient, but returned in a fortnight on account of the right knee, which kept him in hospital for another six weeks, at the end of which time it was, he says, no better.

When admitted to the Leeds Infirmary the knee was swollen, but free from tenderness on manipulation. When the muscles were braced up the bones were in good position, but as soon as the muscles were relaxed the tibia fell backwards until stopped by the ligamentum patellæ. On manipulation the head of the tibia could be brought forward in front of the femur, and there was also very free lateral movement of the head of the tibia on the femur; some fluid in the joint could be felt, and the patella floated. It was manifest that not only were all the ligaments relaxed, but that the crucial ligaments had been ruptured.

Operation (November 21).—The joint was opened by an incision starting at the most prominent part of the internal condyle, and passing downwards and outwards, crossing the ligamentum patellæ about the middle, and extending upwards to the prominence of the external condyle. On opening the joint the synovial membrane was found red and injected, and

there was an excess of synovial fluid. Both crucial ligaments were found completely ruptured, being torn from their upper attachments, the ends being in a somewhat shreddy condition. They were stitched in position by means of catgut ligatures, the anterior being stitched to the synovial membrane and tissues on the inner side of the external condyle; and the posterior, which was too short and was split in order to lengthen it, was fixed by sutures to the synovial membrane and cartilage on the outer side of the inner condyle. The wound was then stitched up by means of buried catgut sutures, and was closed superficially by interrupted silkworm-gut sutures. Some pain followed the operation and there was effusion into the joint, but on removing a stitch a little serous fluid escaped and relieved tension, the after progress being uneventful.

December 4.—Stitches removed. Edges firmly healed; there was still considerable swelling in the neighbourhood of the knee.

December 14.—Plaster of Paris was applied, and he was allowed to get about on a Thomas's splint and to go home. The plaster was removed in a month, after which articular movement gradually returned under treatment by massage.

At my request my house surgeon, Mr. W. Gough, M.B., B.S.Vict., visited him on October 24, 1901, and gave me the following report:—"The patient describes his leg as 'perfectly strong.' He walks well without a limp, and can run. He works eight hours a day at his old employment of 'getting' coal, and has never been off work a day on account of the knee since his discharge from the Infirmary. He has no pain in the knee except when he has overworked it, when he has a little aching over the inner side of the joint. There have been no attacks suggestive of a loose cartilage. On inspection the scar of the old incision is seen. The joint looks fuller than the other and the outlines are rather blurred, the fossæ on either side of the patella being shallower than normal. Measurement showed practically no difference in the circumference of the joints. There is no tenderness over the joint.

"*Movements.*—No abnormal mobility whatever is present. Extension to the straight line is perfectly free. Flexion is somewhat limited, but the knee can be flexed to just beyond a right angle quite freely, when it becomes fixed, and any attempt at further flexion causes pain over the outer side of the joint. On flexion and extension fine creaking is felt in

the joint. The patient says the joint is more liable to pain him in cold, damp weather."

Remarks.—As a rule I do not care for reporting a single example of any disease or injury, but having waited eight years without another case of ruptured crucial ligaments coming under my observation, I think the time has come for the publication of this one, especially as I am able to give a report of the condition so long after operation.

A search through surgical literature has not rewarded me in discovering any other case in which these deeply placed and strong ligaments have been repaired; this therefore will, I trust, be a sufficient excuse for my contribution, as it clearly demonstrates the hopefulness of operation in so serious an injury.

VI.—*On Hepatic Abscess.* By W. K. Hatch, Col. I.M.S.

Read November 28, 1902.

OF late years much has been written on the subject of hepatic abscess, and surgeons who have had experience of tropical diseases have joined in the discussion at the annual meetings of the British Medical Association on the various points connected with the pathology and treatment of this disease; so that most of the profession, even those of us who have never personally treated a case of tropical abscess, are nevertheless well acquainted with all relating to it. As I have had, however, rather exceptional opportunities of seeing and treating cases of hepatic abscess, on the majority of which I have myself operated, I venture to bring before you some of my experiences. Owing to my long connection with the Sir Jamsetjee Hospital, of Bombay, extending over a period of twenty-two years, during which I was first junior, and then senior surgeon in that institution, I have seen either in my own wards, or in those of my colleagues and in private practice, more than 1000 cases of this disease. I have seen in nearly every instance the operations performed by my colleagues at the hospital, and, as before stated, the majority of these cases have been under my personal care. I wish to bring before you a few points in particular as well as explain myself with regard to what I have stated elsewhere, especially in the *Indian Medical Gazette*, as to the dangers of aspiration of the liver; as I gather from what has appeared in the *British Medical Journal* and books that I have not been altogether understood in the matter. Not long since there was a general impression among the medical, as well as among the laity, that abscess of the liver was nearly always met with in Europeans as the result of alcohol, or malaria, or both combined. The table which I now show you will demonstrate that the disease is common enough among the natives, and though there is in a considerable percentage even among natives a history of excessive indulgence in alcohol, yet it seems to me from a careful inquiry in each case that a still larger percentage have suffered from dysentery, and generally severely at some period of their lives. The occurrence of dysentery as well as malaria is, however, so common

among the class of hospital patients that it is by no means easy to say how far either is actually connected with hepatic abscess. But I have seen so many instances in which the attack of dysentery for which the patient actually came into hospital was recent, that the inflammation of the liver ending in abscess, which followed, would certainly suggest cause and effect.

Cases of Hepatic Abscess admitted into the Sir Jamsetjee Jeejeebhoy Hospital, Bombay, from 1878 to 1900.

1884 ... 34	1889 ... 62	1893 ... 48	1897 ... 52
1885 ... 36	1890 ... 52	1894 ... 47	1898 ... 55
1886 ... 38	1891 ... 44	1895 ... 56	1899 ... 95
1887 ... 39	1892 ... 56	1896 ... 47	1900 ... 80
1888 ... 66			

The symptoms of hepatic abscess are so well known that I need only briefly refer to them; there is one, however, which I have not seen mentioned by any author on this subject, and that is the presence of a strong hepatic odour. This symptom can often be easily detected should the patient be in a close, badly ventilated room, on pulling down the bedclothes. Pus in an hepatic abscess has nearly always a strong hepatic odour, and therefore it is not wonderful, especially when the patient is rather emaciated, that the odour should be perceptible. I have frequently had the evidence of my sense of smell confirmed by my colleagues at the hospital, or by the students in the wards. I do not mean to say that this smell is always present, and when an abscess is small this is hardly likely, but occasionally it may be observed, and may even be of importance, as it frequently happens that the formation of an abscess in the liver is so insidious, the temperature being hardly above normal and pain practically absent, that the presence of any additional symptom may be of use. I well remember the case of a medical student in Bombay, who was thought by his medical attendant to be suffering from malarial fever, with slight enlargement of the liver and spleen; his temperature, however, had never been high; he had become much reduced by obstinate diarrhœa. He was living in a small, hot room, and when I approached and removed his clothes for the purpose of examination I at once noticed and remarked on the presence of hepatic odour. This led me to examine and aspirate the liver, with the result that a large quantity of pus was removed. The climate of

Bombay being moist and the temperature over 80° in the daytime, odours of this kind are perhaps particularly noticeable whenever there is no free current of air entering the room.

It also occasionally happens that percussion over an abscess gives a resonant note, and this might appear somewhat perplexing. As the pus from an hepatic abscess is perfectly sweet apart from its peculiar odour, there is no doubt that in this instance either, from proximity and adhesion to the colon, gas has formed or has passed from the bowel into the abscess cavity. On opening such an abscess the pus has been foul-smelling, like that of an abscess situate in the lower part of the abdomen. In a case of this kind I was once able to have a *post-mortem* examination, but no visible opening into the gut could be discovered; the bowel was, however, adherent.

When the abscess is situated near the middle line, in the vicinity of the ensiform cartilage, it sometimes happens that pulsation transmitted from the aorta is plainly noticeable. This may occasion some difficulty in diagnosis, especially if the history be obscure and the temperature little or at all raised above normal. I have operated on several instances of this kind. Again, although the enlargement of the liver is usually fairly uniform, yet it is not uncommon to find increased dulness in the right flank only, posterior to the nipple line, and extending downwards as far as the crest of the ilium, as shown in the diagram. The origin of the abscess might be doubtful, owing to it occupying a position usually filled by tumours, etc., of the kidney.

In one case, which I have published elsewhere, a somewhat movable kidney in close relation with the liver led to my puncturing the kidney, as proved by the presence subsequently of blood in the urine. At the same time there was a very small abscess in the liver, no larger than a hen's egg, which had given rise to serious illness of a month's duration, and which I was fortunate enough to discover and treat successfully. Or, as in the diagram, a long, tongue-like process may extend from the under surface of the liver as far as the pelvis. The patient was an old man, and on examining his abdomen some prominent veins were visible in both iliac regions, and the right rectus muscle appeared slightly bulged. In the right region, between the nipple and anterior oscilliary line, a swelling about two inches square was visible. A sense of resistance was felt on palpation over the abdomen, and in the right hypogastrium a solid swelling could be made out,

and this could be moved by the two hands backwards and forwards. Posteriorly no definite swelling could be found; both regions were tender on pressure.

There was dulness from the right costal angle for an inch and a half downwards, which gradually merged into the impaired note over the swelling, which was not absolutely dull. The edge of the liver could be felt in the costal angle, and one and a half inches below the costal margin. In the course of a few days the abdominal swelling gradually increased downwards into the right iliac fossa, and the dulness above diminished. The temperature rose steadily at night to 100° and 102°. At the end of nine days distinct fluctuation could be felt in the swelling. The patient was therefore operated on that morning. Under chloroform an incision was made from the umbilicus for two inches. On introducing the finger the tumour was felt to have a firm, fine margin on the inner side; on passing the finger upwards the edge could be traced to the liver, near the gall-bladder, which could also be partly seen. It became narrower at this point, and felt firm. A trocar and cannula were introduced about the middle of the swelling; only blood issued. The outer portion was then examined, as it felt softer; the aspiration needle was pushed into it, and pus of a yellow colour passed through. Another incision was now made close above the anterior superior spine, obliquely in direction, and through this the abscess was fairly opened; the pus had an hepatic odour. The tongue-like portion had an appearance of liver, and apparently was an elongated lobulus quadratus. I examined it carefully during the operation, but could not get a *post-mortem* examination on the patient's death, he being a Parsee. The diagnosis made by my friend the late Surgeon-Major Mauser was inflammation in connection with bowels, probably large intestine. It was only at the operation that the nature of the case was clear.

When writing in the *Indian Medical Gazette* three years ago on the subject of aspiration, my object was to bring to the notice of the profession the fact that this operation was attended with a certain amount of danger, and that though an apparently simple proceeding, and one which is generally placed before a patient as a very simple matter, yet it might end in a most unfortunate manner. I also wished to warn those who are, perhaps, rather inclined to use punctures more as an aid to diagnosis than as a necessary procedure when the diagnosis has been more or less accurately established, and it only remains to find the precise locality of the abscess

and to open it in due course. The danger lies either in puncturing an enlarged and highly congested liver, or in introducing the needle directly into some large hepatic vessel; taking into consideration the size of the hepatic veins, it is hardly to be wondered at that this should occasionally occur. Just as one is apt to rely on an exploratory laparotomy for the elucidation of the symptoms, without giving them or the physical signs sufficient amount of consideration, so one is likely to thrust in an exploratory needle in cases in which the liver is enlarged and the symptoms doubtful, without getting beforehand enough evidence of the presence of an abscess. That this is not unattended with danger, even in the hands of careful and experienced men, the following notes will, I think, clearly show; and, moreover, when hæmorrhage has occurred the amount of blood lost may not be very large, but quite sufficient to turn the balance against a patient who has by no means blood to spare. But in other instances the hæmorrhage from this apparently slight operation has been so large as to fill the dependent parts of the abdomen in the course of a few minutes, this being accompanied by rapid blanching of the surface, profuse perspiration, and death within a short period. That the cause of this hæmorrhage has not been puncture of the vena cava, as a speaker on one occasion is reported to have kindly suggested, I have been able to demonstrate both at the time of the operation and by *post-mortem* examination. I might also say that with our large experience of hepatic abscess at the hospital, neither my colleagues nor myself would be likely to wound this vessel either by carelessness or from ignorance of the anatomical relations.

The first case of this kind that I can remember occurred to the late Dr. Vandyke Carter, a careful and able man, as all must admit. He showed me the specimen which he had preserved at the Gowldars Hospital, and also exhibited it to others in Bombay. The aspirating needle had evidently entered into one of the hepatic veins. Some time afterwards I operated myself privately on two patients who were thought to have abscess of the liver by their medical attendants; in one instance free bleeding took place into the bottle, and the patient gradually sank shortly afterwards; in the second the man died some time after our departure, and the symptoms as related to me certainly pointed to death from hæmorrhage. About four years ago a very unfortunate occurrence took place in our hospital practice; two patients were aspirated on succeeding

days by my colleague at the Jamsetjee ; I was present on both occasions, and the needle was introduced in what is usually considered to be a safe position ; no pus was withdrawn, only blood. There was subsequently faintness and symptoms of internal hæmorrhage in both cases. One body was afterwards examined and a large quantity of blood was found in the peritoneum. We could not disturb the parts further, as the patient was a Mohammedan, and owing to recent agitation *autopsy* had been forbidden ; nor could we in the case of the second patient even open the abdomen. But the symptoms were identical ; both men were stout, fleshy, and probably alcoholic. The following case is of special importance and interest because I was myself the operator. In 1897 I was asked by the senior physician at the Jamsetjee to explore the liver of a patient suspected of having an hepatic abscess, as he was expectorating pus of a pinkish colour. He was a big, heavy man, but his muscles were soft and his complexion sallow ; bronchitic sounds were heard all over the lungs ; the liver was enlarged nearly to the umbilicus, and the heart was dilated. Dr. Lyon asked me to aspirate him near the middle line ; under this circumstance I always open the abdomen by a two-inch incision through the rectus and expose the liver, as this region is dangerous owing to the possibility of pushing the needle through the liver and wounding deeper structures. By this method, too, valuable information may sometimes be obtained as to the general condition of the liver, and an abscess may be viewed if near the surface, as a rule, though unless the walls are very thin there is no indication of the presence of pus judging from the colour of the surface. Having carefully exposed the liver by this incision the surface was seen to be extremely dark, of a purple colour and firm feel ; I pushed in a small trocar and cannula as supplied in pocket cases—only dark blood came. I therefore withdrew the cannula slowly, and at once a stream of blood issued from the puncture, which I eventually stopped by steady pressure with my finger. Dr. Lyon considered that there was certainly an abscess in the right lobe. I then entered the aspirating needle, at his request, between the lower ribs—only dark blood came freely. I cautiously withdrew the needle after a few minutes ; the patient became pale and covered with sweat, and his pulse feeble and rapid. Deciding that hæmorrhage must be going on, I rapidly enlarged the incision previously made anteriorly, and passed my hand into the abdomen ; already it contained a considerable amount of blood. Passing

my hand so as to reach the puncture, I pressed upon it until the bleeding had ceased, which it did at once almost, nor did any further bleeding occur from the first puncture. I then removed all blood from the abdominal cavity, but the patient never rallied. The amount of blood lost, though not really large, was sufficient to cause death. This patient was again a Mohammedan, so that we could not ascertain the position of the abscess, which was undoubtedly there, the expectorated pus having an hepatic odour. I was also the operator on the second patient in February, 1897, and I was asked to explore by Dr. Prall. The patient was a butler by occupation, and I fear indulged in liquor considerably. The eyes were jaundiced; the temperature was usually 101° to 103° in the evening; there was considerable emaciation; the spleen could be felt; liver margin extended an inch below the ribs; it was not tender. At the request of Dr. Prall I made two punctures, the second one being rather further back than the first in the same interspace. Only a few drops of dark blood were withdrawn; as I was doubtful myself as to there being an abscess, I decided not to operate further. The puncture was made on February 21, and on the 22nd the man was reported to have slept soundly, which probably meant that the ward boy had done so, as there was no nurse; but he was very feeble, and died at 2 P.M. At the *autopsy* old pleuritic adhesion was found at both bases; the spleen was enlarged and the liver large, fatty, friable, and congested. Beneath the diaphragm was a large clot which extended to the right flank; on removing this about a handful of dark fluid blood was seen to have collected. The track of the second puncture was filled with clot, but on carefully passing a small blunt probe along it was found to lead to a small vein about an inch from its connection with a much larger vessel. There was no abscess. On yet another occasion I was asked to be present when one of the honorary surgeons to the hospital explored the liver. The operation was performed and the patient died next morning. I was present at the *autopsy*, and we found that quite a small vein near the surface of the liver had been transfixed by the needle. There was no abscess; a large amount of blood was present in the peritoneum. I think I have said enough to show that it would be better to resume aspiration for the localisation of the abscess, and not to perform it for merely diagnostic purposes. I am aware that the removal of blood from the liver is often followed by relief to the patient, and sometimes by even com-

plete removal of the symptoms, and that it has been advocated by no less a person than Dr. Harley for treatment of chronic congestion, who considers that it is safe and effective. No doubt one might operate on quite a large number of patients without mishap, but the occurrence of even one result such as I have described would, I think, warrant the greatest caution. Last year's report of Madras Hospital: patient was aspirated and then operated on, irregular rupture found, blood freely oozing; this was plugged and abdomen sponged, liver sutured.

As regards surgical treatment, I consider that incision is undoubtedly the proper treatment, and the earlier the better; it may be advisable in the case of a large abscess to first draw off part of the pus by the aspirator and to open by incision a day or two later, thereby hoping to diminish the size of the cavity and thus lessen the chance of its becoming septic, which is very likely to occur owing to saturation of the dressings or to their displacement, particularly by uneducated patients. If we can find the abscess when it is small we save much destruction of the liver substance, at a time, too, when the patient's strength has not become too much exhausted; it is not, however, easy to localise a small abscess; in searching for one I am of opinion that it is better to remove and again introduce the needle at another place rather than from the original site; there is more likelihood of hitting off the pus. I also prefer to open between the ribs rather than below them; difficulties with the drainage-tube are apt to arise in the latter position. We not infrequently find at *post-mortem* examinations small abscesses that have become encysted, the contents being either fluid or pultaceous. I have never met with any instance in which the symptoms of abscesses, when decided, have passed away, so I am inclined to doubt whether such abscesses ever give rise to any signs of their presence; the history of patients in whom such abscesses have been found may not reveal any former illness connected with the liver; on the other hand, quite small abscesses may give rise to very serious illness with well-marked symptoms. If they can be localised they should be incised. Instead of allowing patients to go on expectorating hepatic pus, an attempt should be made to find and open the abscess by incision. Having frequently operated by removal of portions of the ribs, I find that this is usually quite unnecessary; a sufficiently free incision can be made between the ribs; nor do I think it necessary to excite adhesion or suture the liver before opening the abscess. If the

incision be sufficiently but not too free, the elastic pressure of the abdominal wall seems all that is required to prevent pus finding its way into the peritoneal cavity; only once or twice have I known pus to escape in this direction. In those instances I had made two incisions, in front and laterally, and introduced two large tubes. I suppose that the liver collapsed too suddenly under these circumstances, but no harm resulted. Last year I had a patient suffering from fever and great enlargement of the liver; his illness was of long duration, and he had been tapped twice without success. I could plainly feel fluctuation by bimanual examination. I therefore made a posterior incision, and the patient being young made complete recovery; there was capital drainage. As a rule it is not advisable to open in this position. Curiously enough, small abscesses opened in front through the rectus do remarkably well, in spite of the fact that the opening is not dependent. If the abscess cavity be found on probing larger than at first expected, I usually make another opening after a day or two between the ribs and allow the anterior one to close, gradually diminishing the size of the tube. As syringing the cavity with lotion is not attended with a great amount of success, there is no reason for keeping in both tubes. In fact, I never wash out the abscess unless it has become septic, as shown by the rise of temperature, smell, diarrhœa, red and glazed tongue; then a strong solution of carbolic and other antiseptics must be used, or the patient will certainly die. Early and persistent treatment will usually save the patient. I introduce a large litholapaxy cannula, and inject twice daily with 1 in 40 carbolic acid until the symptoms have subsided.

If a large abscess be allowed to burst of itself, then frequently rapid sloughing of the parts around the opening takes place, until a large open cavity is formed. The drawing which I show illustrates this; the ribs are exposed and an extensive cavity brought to view. After removing the necrosed ribs and packing with gauze wrung out in chloride of lime, I succeeded in very much improving the appearance of the parts, the sloughing ceased, and the patient lived for a fortnight after admission.

In the table it will be seen that the mortality in hospital practice is high; in 1897 it was as high as 97 per cent. In that year the cases were more than usually severe, because patients, on account of the plague, were afraid to come to Bombay, and if they resided in Bombay were disinclined to

enter any hospital owing to the foolish report in the plague hospitals of the doings of the doctors.

The drawings of the two patients here shown will give an idea as to the large size to which abscesses may attain before patients come for treatment. In such cases a fatal issue is certain; still they must be included in the general list. In private practice the mortality is about 20 per cent., owing to early treatment; I cannot, however, prove this by a table. Hepatic abscess is uncommon in females and children. I once saw a lad of nine years with such an enormous abscess that the abdomen was distended as if from dropsy; the amount of pus removed was so large that I hesitate to mention it in case it should be considered a traveller's tale; but dysentery is common enough in children, both native and European.

Time will not allow me to say more on the subject, but for the opportunity given me of reading this paper I beg to offer my hearty thanks.

VII.—*A case of Strangulated Inguinal Hernia, in which "Reduction en masse" was caused by the employment of Taxis.* By Lawrie McGavin, F.R.C.S. *Read November* 28, 1902.

THE patient, a man æt. 52, and a dock labourer by trade, was admitted to the Seamen's Hospital, Royal Albert Dock, on September 20, 1902, with the following history:— For nine years he had suffered from a right inguinal hernia which had always been easily reducible, no signs of strangulation having ever been present. On the night of September 14, however, the rupture suddenly came down and he was unable to get it back. He immediately called in a medical man, who made several ineffectual attempts at reduction by the use of taxis. On the last attempt the mass partially disappeared, and the patient at once experienced great pain and almost immediately commenced to vomit. On the following day the vomit became stercoraceous and continued so till admission, nothing further apparently being done for him.

On admission the patient was evidently *in extremis,* and appeared to be suffering from general peritonitis. His temperature was subnormal and his pulse 115, thready and dicrotic. The abdomen was very distended, and there was some tenderness, especially over the right iliac fossa, but no mass could be felt there. In the right side of the scrotum a soft mass was noticed which, in view of the abdominal condition, suggested collapse of the bowel from perforation, with possibly some omentum still strangulated in the sac. Neither flatus nor motion had been passed for six days; the tongue was dry and brown, the breath extremely foul, and the abdominal type of face well marked. In the hope of being able to do something for what I thought might be a partial reduction of a *hernia en bissac,* with rupture of the bowel at the neck of the sac, I had the patient removed to the theatre. Gas and ether were administered, but before three breaths of pure ether had been taken he suddenly ceased to breathe; artificial respiration was therefore resorted to, but he almost at once vomited a quantity of fæculent matter and died.

At the *autopsy* the following condition was disclosed:— The right side of the scrotum contained a flaccid vaginal hydrocele of moderate dimensions, above which the cord was slightly thickened; there was neither bruising nor ecchymosis of the scrotal contents.

On opening the abdomen some foul fluid escaped. The bowels were distended, and on tracing the small intestine downwards, it led to a point in the ilio-inguinal region, below and rather internal to the tip of the cæcum, where it was found to enter what appeared to be the internal abdominal ring, whose margins were much thickened as from old-standing irritation, and whose aperture measured about an inch in diameter. This was situated over a bulging mass in the iliac fossa, and lay behind and external to the cæcum. On dividing the peritoneum over this mass a sac was reached, which on being opened appeared to be the original sac of the hernia. It contained a portion of the ileum (which measured about ten inches in length) in a condition of extreme congestion, almost gangrenous at the point of constriction, and its mesentery. The site of the strangulation was about a foot above the ileo-cæcal valve. General peritonitis was present, but no actual perforation could be found. The heart was firmly contracted in systole, although the larynx was full of fæcal vomit.

I have thought this case worthy of publication, as much on account of its moral lesson as of its clinical interest, for it serves to emphasise what has been so repeatedly pointed out by many authorities—that the attempt to reduce a strangulation of the intestine by taxis is unwise, and that when persisted in repeatedly such treatment is quite unjustifiable. It is, I believe, unwise to attempt reduction by taxis at all, unless the congestion of the bowel be first to some extent relieved by the application of ice (in cases where it is thought that the vitality of the bowel will not be interfered with by such treatment); and in cases where the vomit is fæculent it is certainly so.

It is, under any circumstances, a step taken in the dark, for unless we can answer certain questions we are obviously exposing the patient to risks of which he knows nothing, and from which we, as surgeons, ought rather to safeguard him. Firstly, is the bowel ulcerated? Secondly, is it adherent to the neck of the sac? Thirdly, can we be certain that there are no adhesions across the neck of the sac, over which we may possibly reduce the contents? And lastly, are we

dealing with a *hernia en bissac?* Again, an element of uncertainty lies in the structural condition of the bowel in question; for just as the skin of one patient differs in texture from that of another, so in one patient the bowels may be thin and semi-translucent, while in another they may be thick and opaque. It is clear that taxis will damage the first more easily than the second under similar conditions. In view of the fact that disasters have happened and will happen again, it is, I think, time to abandon taxis as an empirical measure, unworthy of the name of sound surgery.

Admitting that taxis were a justifiable measure, how long may it be persisted in, and how much force may be exerted? Here are two questions which are obviously without sense, for each depends on the condition of the bowel, which is invisible to the eye, and the second to a great extent on the lightness of touch and the muscular power of the individual surgeon, both of them inestimable quantities, and varying considerably in most of us.

Glancing for a moment at the standard text-books on the subject, we find an attempt made in each to limit the duration of taxis. Thus in one case ten minutes, in another five, in another "a few minutes," and in another three to five minutes are given as the time limit, all of them advising only "a moderate degree of force." Thus the advice given is of a very indefinite order, and leaves much to be desired in the question of standardisation. The most definite opinion of all on the subject is to be found in Messrs. Watson-Cheyne and Burghard's recent work, in which the authors frankly condemn the practice in view of the slight risks of the operation of kelotomy.

Reviewing the dangers of taxis, one cannot help wondering how any surgeon can be willing to face the responsibility of them, for all are serious, and in many the condition of the patient is rendered well-nigh hopeless. In all the mortality is very high. Thus "reduction *en masse*" has a mortality of 57 per cent. for all herniæ (inguinal 50 per cent., femoral 73·6 per cent.);* resection of small intestine for gangrene, 50 per cent.;† suppurative peritonitis (in cases operated on), 60 per cent.‡

There must be considered also the possibility of asphyxia from fæcal vomiting if the case eventually comes to operation

* *Guy's Hospital Reports*, vol. lvi, p. 149, Rowlands.
† *New York Med. Journ.*, 1889, McCosh.
‡ Gould and Warren, *International Text-book of Surgery*, vol. ii, p. 324.

(as in the case I have reported), of septic pneumonia, and even (in the highly improbable event of spontaneous cure) of the occurrence of an intractable fæcal fistula. Against these heavy odds must be placed the post-operative mortality of kelotomy, which, if performed within twenty-four hours, stands at 10 per cent. to 12 per cent., but if over three days from the time of strangulation, at 50 per cent.*

While I personally do not recognise the position of taxis under any circumstances as a sound surgical measure for the relief of strangulated herniæ, I am fully aware that there are many surgeons who still admit its utility. I would therefore, in deference to these authorities, suggest the following conditions as being those in which such treatment is absolutely contra-indicated, and it will be seen that the margin in which cases suitable for treatment by taxis may be found, is a very narrow one:—(1) All femoral cases, for those are usually found in women whose intestines are thin, the bowel commonly tightly constricted, and lying on the knife-edge of Gimbernat's ligament in a canal whose peculiar shape renders reduction somewhat difficult at all times. (2) In all inguinal and umbilical cases when (*a*) the strangulation is more than twelve hours old; (*b*) the hernia has long been irreducible; (*c*) the vomit is fæculent in the least degree; (*d*) the hernia is a large scrotal one of old standing with a tight, rigid ring, such herniæ, as Rowlands points out,† being those which are most prone to "reduction *en masse*;" (*e*) any sign of inflammation is present; (*f*) pain is a marked symptom; (*g*) the attempt has already failed.

"Reduction *en masse*" is a comparatively rare occurrence, and Rowlands,† in his recent investigations on one hundred cases, points out that at Guy's Hospital only fifteen cases have been recorded in the last forty years, the subperitoneal reduction into the iliac fossa being the usual variety. In my own case the chief points of interest are, I think, the large size of the mass reduced, the entire absence of damage to the contents of the scrotum, the question raised by the death of the patient as to the advisability of washing out the stomach prior to operation, and that of the best method of dealing with these cases.

With regard to lavage, Treves ‡ has recommended this plan, and the soundness of it must be obvious to all surgeons;

* Gould and Warren, op. cit., p. 466.
† Rowlands, op. cit.
‡ *Manual of Operative Surgery*, vol. ii, p. 383.

for in urgent cases the immediate danger of suffocation under the anæsthetic is considerable, and in the post-operative treatment the risk of septic pneumonia is reduced, as is also the danger of absorption of fæcal material from the stomach. A point of importance is, I think, that if it is done at all it should be done previous to the administration of the anæsthetic, in view of the possible embarrassment to an already feeble respiration. In the present instance I did not consider it wise to attempt lavage, owing to the extreme urgency of the case. As to the method of operating in these cases, the idea of performing a laparotomy before investigating the state of the bowel at the seat of constriction, through the inguinal canal, seems to me dangerous, from the possibility of rupturing it at a weak spot in the attempt to withdraw it from the sac. That such operations are successful is evident from the fact that Erichsen* mentions three cases, two by Thorburn and one by Barker, which resulted in cure; but when the diagnosis is in doubt, or, as in the case recorded by Rowlands in his paper,† where perforation into the sac has actually taken place, the performance of laparotomy must be an extremely dangerous practice.

* *The Science and Art of Surgery*, vol. ii, p. 845.
† Rowlands, op. cit.

VIII.—*Five cases of Intestinal Perforation in Enteric Fever treated by Operation.* By H. J. Waring, M.S. *Read December* 14, 1902.

During the past four years I have had the opportunity of operating upon five patients suffering from enteric fever in whom perforation of the intestine had occurred. Two of the cases were followed by complete recovery, and three terminated fatally. I have brought forward the series of cases because they illustrate many of the varieties and complications which may be met with in the surgical treatment of this class of patient, and also to place them on record, since I think it is advisable when new and improved methods of treatment are adopted that every case so treated should be published. This allows reasonable conclusions to be drawn by making an investigation of the literature of the subject and collecting groups of cases.

Case 1.—A somewhat emaciated, unhealthy-looking schoolboy, æt. 10, was admitted to St. Bartholomew's Hospital on what was considered to be the sixth day of an attack of enteric fever. For eight days following admission the disease appeared to be taking an ordinary course, when without any apparent cause the patient had an attack of abdominal pain. The pain seemed to be general, and not located in any special part of the abdomen. At this time the temperature was 99°, and the pulse 80. Previously the temperature had fluctuated between 102° and 104° F. Three hours later the patient complained of acute abdominal pain, and vomited four times a quantity of bile-stained fluid. On abdominal examination there was general tenderness and rigidity of the abdominal muscles. *Per rectum* there was swelling and tenderness in the recto-vesical pouch. The temperature was now 103° and the pulse 100. One hour later the abdomen was more rigid and somewhat retracted, and only moved slightly on respiration. The temperature had fallen to 99°, and the pulse increased in frequency to 120. A perforation of the intestinal canal was diagnosed, and Dr. Norman Moore, the physician in charge of the case, sent for. Dr. Moore, on his way to the hospital,

called on me and asked me to go down in case an operation was necessary. When seen by Dr. Moore the local condition was practically unchanged, the temperature was 99·6°, and the pulse 170, small, hard, and irregular. The diagnosis of perforation was confirmed and immediate operation decided upon. Abdominal section was performed six and a half hours after the attack of severe abdominal pain, or nine and a half hours after the first onset of acute abdominal symptoms. An incision was made three and a half inches long below the umbilicus and to the right of the middle line. A quantity of gas and some yellow, viscid fluid were found in the peritoneal cavity. The lower portion of the ileum was at once examined and a small perforation found about twelve inches above the ileo-cæcal valve. The perforation was circular and about one quarter of an inch in diameter. This was at once closed by the insertion of six fine silk sutures, each introduced by Lembert's method. It was found necessary to place the sutures wide of the opening owing to the fact that the perforation was in the middle of a rather large ulcer. The exposed part of the peritoneal cavity and the bottom of the recto-vesical pouch were well sponged out with a solution of mercury biniodide 1 in 4000. A drainage-tube was passed to the bottom of the recto-vesical pouch and the greater part of the parietal wound closed. A Keith's dressing was then applied. Considerable difficulty was experienced in feeding the patient, and for the first few days nothing but nutrient enemata could be given. The parietal wound suppurated in its superficial part; otherwise no further difficulty was experienced beyond the occurrence of crops of boils in the dorsal region. Ultimately the patient recovered after a somewhat tedious convalescence. On discharge he had a ventral hernia at the seat of the parietal incision, but when seen two months later this was improving.

Case 2.—The second case was that of a housewife, æt. 29. She was admitted to the Metropolitan Hospital on what was thought to be the tenth day of an attack of enteric fever. She had a severe attack of the disease, intestinal hæmorrhages occurring on the sixteenth, nineteenth, and twentieth days. Afterwards she began to improve, the temperature became almost normal, and convalescence appeared to be established, when she was given solid food. Two days later, the forty-second day of the disease, she began to complain of pain in the right side of the abdomen. This became rapidly worse, vomiting occurred

at intervals, and the temperature rose from 98° to 103°, and the pulse from 80 to 118 in four hours. Perforation of the intestine was diagnosed, and an operation decided upon. This was performed eight hours after the onset of the acute abdominal pain. At the time of operation the abdomen was distended, rigid, and extremely tender to the touch, and scarcely moved during respiration. The pulse could scarcely be counted, and the temperature was 103°. Pelvic examination did not yield any definite information. A median subumbilical incision was made and the intestine investigated. A portion of the small intestine about four inches above the ileo-cæcal valve was found to be slightly adherent to a piece of the great omentum and the deep aspect of the anterior abdominal wall, and covered with flakes of fibrinous lymph. On removal of this lymph by sponging it was found to cover a large ulcer in the wall of the intestine, which had become perforated. The parts were carefully sponged clean and the aperture closed by the insertion of four fine silk sutures after Lembert's method. During the insertion of the sutures a second ulcer, close to the first, was made to perforate. This necessitated the introduction of a second tier of sutures. A quantity of lymph was found on the surface of some of the coils of intestine, and in the recto-vesical pouch. This was removed and the entire area sponged with a solution of mercury perchloride 1 in 4000. The wound in the parietes was then completely closed. The day following the operation the temperature became lower, the pulse remained the same, and occasional attacks of vomiting occurred. These, however, soon ceased, and the patient made a slow but continuous recovery.

Case 3.—The third case was that of a man æt. 39. He was admitted to the Metropolitan Hospital on account of an attack of enteric fever on what was considered the ninth day of the disease. The patient had a severe attack of the disease: he suffered much from sleeplessness, and had intestinal hæmorrhage on the eighteenth, twentieth, and twenty-second days. On the twenty-third day he complained of severe pain in the right iliac fossa. There was rigidity of the abdomen, vomiting, but no marked change in the temperature or pulse. Later in the day the temperature dropped suddenly to normal. There was profuse sweating, and the patient became collapsed. During the night the condition remained the same, but in the morning of the twenty-fourth

day vomiting occurred, the general condition was much worse, whilst the face assumed a pinched and dusky appearance. Peritonitis was diagnosed, and I was asked to see the patient with a view to operation. The patient then had undoubted peritonitis, and his condition was serious. A median incision was made below the umbilicus and the abdomen explored. Free gas and a quantity of purulent matter were evacuated from the peritoneum, and then the intestine was examined. A perforation was at once found in the ileum at a spot about twelve inches above the ileo-cæcal valve. This was sewn up with fine silk sutures. The peritoneal cavity was then sponged free of fluid and a quantity of hot saline solution introduced. The parietal wound was then closed. The patient recovered from the anæsthetic and rallied; then he sank and died six and a half hours later. The operation was performed thirty hours after the occurrence of the acute attack of pain in the right iliac fossa.

Case 4.—The next case is that of a boy æt. 16, who came under observation on the seventh day of an attack of enteric fever. The patient was seriously ill and suffered from a severe attack of the disease. During the entire period he had continuous diarrhœa, which did not respond to treatment. On the twenty-first day of the disease the patient had a rigor, the temperature rising to 103·6° and the pulse to 140. Four hours later he complained of severe pain in the abdomen, especially in the right iliac fossa. On examination the abdomen was found to be distended and tympanitic, and there was marked tenderness in the right iliac fossa and the adjacent part of the hypogastrium. The condition became worse on the following day and continued until the morning of the twenty-third day, when I was asked to see the patient. The abdomen was then distended and tympanitic; there was abdominal pain and tenderness, especially in the right inguinal and hypogastric region, whilst the hepatic dulness had entirely disappeared. Perforation of the intestine with free gas in the peritoneal cavity and acute peritonitis was diagnosed, and an immediate operation decided upon. The abdomen was opened by a subumbilical incision. Free gas and fæcal smelling fluid were found in the peritoneum. These were evacuated, and the lower part of the ileum, the region of the vermiform appendix, and the lower portion of the ascending colon and cæcum investigated. No perforation could be seen. The sigmoid flexure was then sought for and

drawn into the wound. On examination two perforations were found in it, one the size of a pea and the other a little smaller. Each aperture was closed by Lembert's sutures of fine silk. The recto-vesical pouch and the exposed part of the peritoneum were sponged dry, and a large gauze drain passed down to the bottom of the pelvis. The parietal wound was partially closed. In this case perforation probably occurred thirty-eight hours before the operation was performed. The patient rallied for a time after the operation, and took and retained food and stimulants by the mouth. On the following morning, however, he got worse, and succumbed twenty hours after the operation.

Post-mortem.—General peritonitis was found, whilst the lower part of the ileum and the large intestine down to the anal margin were the seat of numerous enteric ulcers. The ulcers which had perforated were situated sixteen inches above the anal margin.

Case 5.—My fifth patient was a young man æt. 21, who first came under observation on the twenty-first day of his disease. He appeared to be suffering from a severe form of enteric fever, and was stated to have had intestinal hæmorrhages on the eighteenth, nineteenth, and twentieth days. On this day (the twenty-first) he was suddenly seized with severe abdominal pain, which was located by the patient in the region of the right iliac fossa and the adjacent portion of the hypogastrium. The abdomen soon became rigid and tender all over. The temperature fell at first and then rose, whilst the pulse was very quick and almost imperceptible at the wrist. A diagnosis of perforation of an enteric ulcer of the intestine was made and an immediate operation decided upon. This was performed six hours after the occurrence of the severe abdominal pain. A median subumbilical incision was made and the peritoneal cavity opened. General peritonitis was found in the area of operation, and a small amount of purulent matter in the recto-vesical pouch. This was removed by sponging and the small intestine in the region of the ileo-cæcal valve examined. Two perforations were found, one five inches above the ileo-cæcal valve and the other two inches higher. Each aperture of perforation was more than a quarter of an inch in diameter, and had thin, undermined edges. These apertures were closed by a series of fine silk sutures, each introduced by Lembert's method. Adjacent parts of the intestine were examined for further

perforations, but none found. The recto-vesical pouch and the peritoneum and coils of intestine within the area were then sponged with a solution of biniodide of mercury, a drainage-tube passed to the bottom of the pelvis, and the upper two-thirds of the abdominal incision closed. The patient improved for about twelve hours after the operation, then began to sink, and died twenty-four hours later.

Post-mortem.—Acute general peritonitis was found in the lower half of the abdomen, whilst within the intestine very extensive ulceration was found in the lower part of the ileum. One ulcer was apparently on the point of perforating.

The most noteworthy points in connection with this series of cases are—

1. *The seat and character of the perforation.*—In four of my cases the perforation was situated in the lower twelve inches of the ileum (4, 5, 12, and 12 inches respectively), and in the fifth case in the lower portion of the sigmoid flexure sixteen inches above the anal margin. Two of the cases had a double perforation, and in each case the perforations were close to one another. This observation is in agreement with other statements which have been made on the subject. The practical deductions to be drawn from this are that when an operation is performed the lower one or two feet of the ileum should be first examined, next the region of the cæcum, vermiform appendix, and the lower portion of the ascending colon, and then the sigmoid flexure. An examination of all the recorded cases shows that more than 90 per cent. of the perforations have been found in one of the three regions mentioned. As regards the method of suturing, I have in all cases used fine silk, each suture being introduced by Lembert's method. Catgut I do not think will be found suitable for the purpose, owing to its thickness and the short time it takes to be absorbed. When introducing the sutures care must be taken not to perforate an adjacent ulcer. In one of my cases this accident occurred. The position of other ulcers can generally be recognised by the areas of subperitoneal redness which overlie the ulcers. I do not think it advisable to attempt to excise the ulcer, on account of the probability of invading other ulcers, and also on account of the extra time required for the operation and the increased shock to the patient.

2. *The most suitable period for operation.*—My operations were performed 6, 8, 9½, 30, and 38 hours after the occurrence

of perforation, as far as could be judged from the clinical histories of the patients. The patients who recovered were those at 8 and 9½ hours; the fatal ones 6, 30, and 38 hours from the time of perforation. It would thus appear that the sooner an operation is performed after the occurrence of perforation the greater are the patient's chances of recovery. When time is lost owing to difficulties in making a diagnosis or in obtaining the services of a surgeon, the greater are the probabilities of the establishment of acute general peritonitis. Four of my series undoubtedly had acute peritonitis in the recto-vesical pouch and the lower part of the peritoneal cavity. Three of these died and one recovered. In the remaining case there was fibrinous lymph on the surface of the omentum and several coils of intestine, but only a small amount of liquid exudation. Some surgeons have stated that the most favourable period of operation is between the twelfth and twenty-fourth hour. My cases are not sufficient to controvert this view, but I feel strongly from what I have seen in my own cases, and also those I have seen at St. Bartholomew's Hospital, that the sooner an operation is done the greater are the chances of recovery.

3. *The period of the disease at which the perforation takes place.*—Published accounts of cases seem to show that the later the stage of the disease the greater are the chances of an operation being successful. It appears to me, however, that success depends mainly upon the general condition of the patient at the time of perforation, the effects the disease has produced upon his vitality, and the early performance of an operation. In my successful cases perforation apparently took place on the fourteenth and forty-second days; and on the twenty-first, twenty-second, and twenty-third days in the fatal ones. As a help in establishing a correct diagnosis in doubtful cases in which enteric fever undoubtedly is present, but the symptoms of perforation are indefinite, I think that systematic examination of the blood ought to be made in order to detect leucocytosis if this is present. When leucocytosis is found, and there are no signs of suppuration in any other part of the patient, it is probable that there is peritonitis, caused by a perforation of one of the intestinal ulcers, and an operation ought to be carried out without delay. In enteric fever there is usually no leucocytosis, unless peritonitis is present, or suppuration is established in a mesenteric gland, in a joint, or in connection with bone.

4. *Results.*—My five cases show 40 per cent. of re-

coveries and 60 per cent. of deaths. Two of the cases terminated fatally, I think, owing to the length of time which was allowed to elapse between the first signs of perforation and the performance of an operation. The other fatal case was one of a very severe form of enteric fever, and, if the operative procedure had been successful, another ulcer was on the point of perforating.

Conclusions.—The conclusions I have drawn from my cases are—

(*a*) That all cases of perforation of the intestine in enteric fever ought to be submitted to operation as soon as possible after the perforation has occurred.

(*b*) That the operation ought to consist in abdominal section, careful suture of the perforation with interrupted fine silk sutures, and drainage of the recto-vesical pouch. The recto-vesical pouch should first be sponged free of all lymph and exudation and then wiped with a sponge soaked in a solution of biniodide of mercury 1 in 4000. When there is much collapse the recto-vesical pouch may be filled with hot sterilised normal salt solution. One of my patients was much improved at first by this proceeding.

(*c*) When the operation is being performed the lower portion of the ileum should be examined first, next the region of the cæcum and vermiform appendix, and afterwards the sigmoid flexure. If nothing is found in these regions, then the gall-bladder and the lymphatic glands in the mesentery should be investigated. Cases have occurred in which all the symptoms of perforation have been caused in the course of enteric fever by the rupture of an empyema of the gall-bladder or a suppurating mesenteric gland.

IX.—*A case of Typhoid Fever; Perforation during a relapse; Laparotomy; Death on the third day from a second Perforation.*

By J. Walter Carr, M.D., and Edmund W. Roughton, B.S. *Read December* 12, 1902.

Florence B., æt. 18, a factory hand, was admitted to the Royal Free Hospital on October 7, 1902, on what was probably the ninth or tenth day of an attack of typhoid fever. The previous history was unimportant. The disease was evidently of a severe type; the temperature rose to 105° on the first two days after admission, but was controlled by sponging, so that it did not afterwards reach 104°, and seldom above 103°. Before admission there had been constipation, but the administration of two grains of calomel was followed by considerable diarrhœa, which, however, soon subsided under starch and opium injections.

During the first ten days there was nothing to note of special importance, except that all the symptoms pointed to the serious character of the disease; the tongue showed a marked tendency to become dry, and sordes rapidly accumulated on the lips and teeth; the pulse was small and frequent, varying between 120 and 130, and the heart's apex-beat became almost imperceptible; there was a good deal of bronchitis, and the abdomen was always considerably distended; vomiting was a troublesome symptom throughout, and there was marked muscular tremor, suggestive probably of deep intestinal ulceration. The patient, however, continued to take food fairly well, had very little delirium, and never passed into a typhoid state.

On October 18 she had a small intestinal hæmorrhage, not sufficient to cause any marked collapse, though the temperature fell abruptly from 103° to 99°. As not uncommonly happens in such cases, this was followed by a distinct improvement in the patient's condition; she became brighter and more rational, and it is particularly worthy of note that although before the hæmorrhage she had been taking brandy, which had been gradually increased up to 6 oz. *per*

diem, and afterwards this was entirely omitted, yet the pulse certainly did not get weaker or more frequent, and the tongue became slightly moister.

Towards the end of the fourth week of the disease the temperature began to fall, but was never normal for the whole twenty-four hours; during the fifth week it rose again, and a few days later fresh spots appeared, indicating the probable occurrence of an intercurrent relapse. The temperature now varied between 101° and 103°, and the strain of the long-continued fever was shown by a gradual rise in the pulse-rate to 140, and in the respiration rate to about 40 per minute. However, the patient's condition continued to be fairly satisfactory until November 6 (about the fortieth day of the illness), when about 4 A.M. her temperature rose to 105·2° and the pulse to 160; there were no signs of peritonitis, the abdomen was not tender or rigid, and moved freely with respiration; after sponging and a hypodermic injection of gr. $\frac{1}{3}$ of morphine, her condition improved and she slept quietly. At 9 A.M. she woke with violent abdominal pain; the temperature had fallen to 101°, the abdomen was much distended, rigid, and very tender, and the liver dulness absent. It being obvious that perforation had occurred, preparations were at once made for operation. This was commenced at 9.55 A.M. An incision four inches long was made an inch to the right of the middle line; the anterior sheath of the rectus was incised, the muscle displaced outwards, and then the peritoneal cavity opened. Much gas escaped. The perforation was at once found. It was circular, clean-cut, and about $\frac{1}{16}$ inch in diameter. The adjacent coils of intestine were soiled with a greenish-yellow fluid, looking more like bile than ordinary typhoid stool. The perforation was quickly and easily closed with a double row of Lembert's sutures. The soiled intestines were carefully sponged with plain gauze. No washing or irrigation was used. The abdomen was closed with three rows of sutures. No drainage of any sort was employed. The usual dressings were applied and the patient was sent back to bed at 10.20, the operation having occupied nineteen minutes.

Her condition at the end of the operation was if anything somewhat better than at the commencement; she recovered from the anæsthetic without vomiting and retained two nutrient enemata, one given immediately after the operation, and the second at 2.30 P.M. Strychnine and digitaline were administered hypodermically. At 5.30 P.M.,

as the pulse was becoming feebler, feeding by mouth was resumed, small quantities of raw meat juice in iced water, and of peptonised milk, being given alternately every half-hour.

During the next forty-eight hours or so patient's condition was fairly satisfactory; the temperature varied between 99° and 102°, and the pulse-rate was about 140. She took food well, but was slightly sick at times, though not more than before the operation. The bowels were open twice, and flatus was passed freely; there was no complaint of abdominal pain. The abdomen was soft and moved well with respiration.

During the evening of November 8 the temperature rose to 103°; shortly afterwards, at 9 P.M., patient complained suddenly of severe abdominal pain. The abdomen was found to be distended, tender, rigid, and tympanitic, with loss of liver dulness. Patient rapidly sank and died about midnight, sixty-two hours after the laparotomy.

Permission was only obtained to examine the abdomen through the operation wound. The perforation, which was closed at the operation, was found to be situated a little over twelve inches above the ileo-cæcal valve; it was firmly sealed, and there was no peritonitis round it. Five inches or so lower down a second perforation was found, about half the size of a threepenny-bit; this was evidently the cause of death, as there was fæcal matter and free gas in the peritoneal cavity, though the peritoneum itself was only slightly injected. There were several ulcers, the floor of which was only formed by peritoneum, and some of these would most probably have perforated had the patient lived longer. Other ulcers were apparently healing, and were presumably those which had formed during the primary attack.

Remarks.—The rise of temperature which distinctly preceded the definite signs of perforation in each instance is worthy of note as a somewhat unusual symptom, and yet perhaps suggestive of imminent perforation. The chief interest of the case, however, depends on the comparative success of the operation for the first perforation. From the appearance of the parts at the *post-mortem* examination, there would seem to have been very little danger of the supervention of peritonitis. Shock was completely recovered from, and as the patient's condition twenty-four hours after the operation was not materially worse than it had been twenty-four hours before it, it is possible, if not probable, that, but for the occurrence of the second perforation,

recovery might have followed, although no doubt the general condition was sufficiently grave.

The case, then, is of value as showing the possibility of a good result following the suture of a ruptured typhoid ulcer, even under the most unfavourable conditions; for it should be borne in mind that not only did the perforation occur—as must, of course, usually be the case—whilst the disease was in the acute stage, but after it had lasted uninterruptedly for nearly six weeks, with severe and even dangerous symptoms throughout, so that the patient was naturally in the worst possible condition for recovering from an operation. On the other hand, certain favourable factors must not be overlooked: first, and most important of all, the performance of the operation within an hour or so of the onset of definite symptoms of perforation, thanks mainly to the promptitude with which the house physician, Miss Louisa Hamilton, M.B., recognised the condition and sent for surgical help; secondly, the exceedingly small size of the perforation, in consequence of which very little fæcal matter had been extravasated, although gas had escaped freely and so facilitated an early diagnosis; thirdly, the discovery of the perforation immediately upon the opening of the abdomen, so that the operation was rendered a comparatively short one, and consequently the amount of shock greatly diminished. The morphine, which had been given a few hours before, also probably helped to lessen shock and prevent much fæcal extravasation.

In the great majority of cases of perforation in typhoid fever it would be unreasonable to expect such a combination of favouring circumstances.

Lastly, the final result naturally raises the important question, in what proportion of typhoid fever patients who perforate during the acute stage is a second perforation likely to occur, supposing the dangers of the first are averted? Probably further *post-mortem* evidence is desirable on this point, as it is one which has only become of practical importance within the last few years, since the performance of laparotomy in cases of perforation has been considered justifiable. A second operation is not likely often to be attempted within a few days of the first, and in the present case it was felt that the risk of the patient dying on the table was too great to allow of anything further being done, and the rapid way in which the patient collapsed fully justified this decision, although a patient in America recovered after being operated upon three times in a fortnight—twice for perforation and

once for a supposed perforation. Mr. Parkin, of Hull, has recorded (*Brit. Med. Journ.*, vol. i, 1895, p. 192) a very similar case to our own: symptoms of perforation occurred about the end of the third week of an attack of typhoid fever; the abdomen was opened two hours later, and a perforation about the size of a pin's head found and closed. The patient made a good recovery from the operation, but died three days later with symptoms pointing to a fresh perforation, except that, instead of a fall of temperature, there was a rapid rise. At the *autopsy* no peritonitis was found round the sutured gut, but another ulcer was on the point of perforating, and around it there was evident peritonitis.

When an ulcer perforates during convalescence or in the course of a mild attack, the danger of a second perforation is necessarily very much less, and it is in such cases that most of the successful results hitherto recorded have occurred, at any rate in this country. So far as we can ascertain, seven cases of recovery after operation for perforated typhoid ulcer have up to the present been recorded in the United Kingdom (particulars are given in the annexed table; cases of merely localised peritonitis or abscess are not included). Three were quite convalescent from the fever, and in each of the other four the attack was a mild one and the patient's condition satisfactory when the perforation occurred. In other words, whilst the great majority of perforations occur in cases of a severe type, and whilst the patient is still seriously ill, not a single successful operation under these circumstances has yet been recorded in this country.

Cases recorded in the United Kingdom of recovery after operation for perforating typhoid ulcer.

No.	Recorded by—	Date.	Reference.	Patient's condition when perforation occurred.	Probable interval between perforation and operation.	Duration of operation.
1	Sir Lauder Brunton and Mr. Bowlby	Dec., 1895	*Med.-Chir. Trans.*, vol. lxxx	Quite convalescent . .	? 18 hours	40 minutes.
2	Dr. Handford and Mr. Anderson, of Nottingham	March, 1898	*Brit. Med. Journ.*, vol. ii, 1898, p. 220	Attack never very severe; perforation on 33rd day, when temperature had been normal for nearly a week, and patient was "quite convalescent"	22½ hours	?
3	Mr. Platt, of Manchester	Nov., 1898	*Lancet*, vol. i, 1899, p. 505	Perforation on 13th day of a mild attack; hardly any fever for several days after operation, but a relapse subsequently	22 hours	50 minutes.
4	Dr. T. H. Dalziel, of Glasgow	Nov., 1898	*Scottish Med. and Surg. Journ.*, vol. ii, 1899	Only scanty details given, but case apparently mild, and temperature normal before perforation occurred	11 hours	28 minutes.
5	Dr. Marsden, of Manchester	Jan., 1900	*Lancet*, vol. i, 1900, p. 1800	Perforation near end of a relapse of moderate severity, when patient's general condition was "extremely satisfactory"	8½ hours	30 minutes.
6	Mr. Henston, of Dublin	Sept., 1900	*Brit. Med. Journ.*, vol. ii, 1901, p. 1454	Patient's temperature had been quite normal for four or five days	4 or 5 hours	?
7	Dr. Malet and Dr. Deansley, of Wolverhampton	Feb., 1901	*Brit. Med. Journ.*, vol. i, 1901, p. 1079	Perforation on 15th day of a mild attack, when patient's condition was steadily improving, and morning temperature had become normal. Scarcely any fever after operation	7 hours	About one hour.

X.—*A Second case of Successful Operation for Perforation in Typhoid Fever.* By Anthony A. Bowlby, C.M.G. *Read December* 14, 1902.

THE patient was a boy æt. 10, who was admitted into St. Bartholomew's Hospital under the care of Dr. Norman Moore on September 1, 1900. He was in the third day of an attack of typhoid fever, and beyond the fact that the case was severe and prolonged it called for no special comment, except that after the temperature had come down to normal on the thirteenth day he suffered from a severe relapse on the twenty-first day, and had again a very high temperature, reaching 105·4° F. on one occasion. The date of the relapse was September 19. On October 1 the temperature was still above normal (101°), and tympanites, which had been present throughout, was still well marked. There was also some crepitation at the bases of the lungs. On the 2nd, after a slight shivering fit, the patient became blanched and cold, the temperature sinking to 97·6°, but there was no subsequent evidence of hæmorrhage or pain. On the 3rd he seemed to be better, but the temperature was still 101°. The abdomen was less distended and the bowels acted without enemata. On the 4th the temperature was normal, but the abdomen was again distended. The pulse was 96. A little before 3 P.M. he complained of some difficulty in micturition, and while passing urine he was seized with severe pain in the abdomen. The temperature half an hour earlier had been 102·6°, but after the attack of pain the patient sweated profusely, and it dropped to 98°. For the next half-hour the boy was screaming with pain and was exceedingly restless. He then became quieter and collapsed, with sunken eyes, a cold sweat, and shivering. The pulse had gone up meantime to 140. I saw him at 4.30 P.M. with Dr. Norman Moore, and found his general condition to be as above described. The abdominal wall was hard and fixed during respiration, and I agreed with Dr. Norman Moore that perforation had almost certainly taken place, and quite concurred in his proposal that an operation should be undertaken at once. At 5 P.M. therefore, two hours after the symptoms of perforation, the

abdomen was opened in the middle line, and at once free gas was found in the peritoneum. A considerable quantity of almost clear fluid welled up, but there was no large collection of fæcal matter. The intestines were so much distended that several coils had to be allowed to extrude, and they were kept wrapped in hot towels. Many areas of inflamed peritoneum indicated the affected Peyer's glands. I then sought for the cæcum, and after finding it I rapidly passed the small intestine through my hands till I found a perforation situated less than two feet from the ileo-cæcal valve. The bowel was not adherent or otherwise altered, nor could any induration be felt in its walls, but the peritoneum corresponding to the shape of a Peyer's patch was very red and injected. The opening in the intestine was quite small, being not much larger than a pin's head. I wiped the bowel clean and then closed the opening by five Lembert's sutures of fine silk. The greater part of the incision was now closed, and a glass drainage-tube was placed in its lower end. Owing to there being no complications the operation was completed in about twenty minutes, and at the end of it the patient's general condition was none the worse for the handling and exposure of the bowel. The peritoneum was not douched, but the intestines were carefully wiped clean and all fluid was completely mopped up from the pelvis and elsewhere with sponges. For several days the boy remained very ill, and in the early morning after the operation the pulse was still 150, the temperature being 100·4°. There had been no sickness, and flatus was passed. The patient was fed by nutrient enemata, and only a little water or ice was given by the mouth. The abdomen remained distended. After this time there was steady and rapid improvement except for some sickness on the 7th and a troublesome cough, so that by the 10th (six days after the operation) the boy seemed to be convalescent. On that day, however, he had a slight epileptiform attack, and in the evening a more severe series of fits, necessitating the administration of chloroform. This threw him back and weakened him, but he again rallied, and, in spite of a return of severe convulsions on the 20th, he ultimately recovered. The tympanitic distension of the abdomen which had been so marked throughout continued till the end of his illness. I saw the boy some months later, and found him to be in excellent health.

I have already recorded a similar case of operation for perforation of typhoid ulcer in vol. lxxx of the 'Transactions'

of the Royal Medical and Chirurgical Society (in conjunction with Sir Lauder Brunton), and in that case also, which had a similar satisfactory ending, the patient was operated upon late in an attack of typhoid fever, for he was in the sixth week of the illness and was at the time suffering from a relapse. In that case there was much induration of the perforated Peyer's patch and some consequent difficulty in suturing the aperture, but in all other respects the cases had much in common except that at the time of the operation the boy whose case I now record was much more ill than was the first patient, and that there was greater distension of the abdomen.

The two cases represent the only patients I have operated upon, and there can be no doubt that they represent two lives saved. On the other hand, however, I do not regard them as at all typical cases of typhoid perforation, but rather as typical of a special class in which perforation occurs after the fever is practically over, and in which the general condition of the patient is otherwise relatively good. In such patients as these the symptoms of perforation are generally well marked, and the diagnosis is readily arrived at. But in very many other cases the reverse is true, for it not infrequently happens that the perforation occurs when the fever is yet high in patients who are nearly or quite unconscious, and in whom there is no perception or expression of pain, and who may have distended and tympanitic abdomens before perforation occurs. Many such patients are already at death's door, and in not a few of such cases, in addition to the perforation, there are numerous other areas where the intestine is so rotten that its giving way is only a question of time. It is not to be supposed that even if an ulcerated bowel is sutured many such patients can recover, and in some of them at least the operation of suture may be practically impossible. I saw at least one such case in South Africa with Dr. H. H. Tooth, in which the patient was quite unconscious and manifestly dying. Dr. Tooth suspected that there might be a perforation, but as the symptoms were not clear no operation was undertaken, and the same day the man died. A *post-mortem* examination showed that the intestine was so matted and adherent to the peritoneum and mesentery that even after death it could with difficulty be unravelled; and though there was a perforation I do not think that it would have been discovered by operation, even if the patient's condition had permitted one to be performed.

As surgeons we are of course entirely dependent on physicians for the opportunity of operating on cases of perforation in typhoid fever, and it is evident that it is for us to convince them that operations may save life. The case which I have recorded is one in which the saving of life was primarily due to Dr. Norman Moore, and in recording it I do not wish to suggest that all cases are likely to be so successful. On the other hand, I do think that in the present position of intestinal surgery there is good reason to advocate that the chance given by operation should not be withheld in any case of perforation, and I would suggest that in all such cases where perforation is suspected a surgeon should be called in as promptly as possible.

XI.—*On the Subcutaneous Injection of Paraffin for the treatment of Sunken Noses.* By Stephen Paget. *Read December* 14, 1902.

I VENTURE to submit to the Society some practical experiences of this method. There is no time to go into the history of it; but I would remind the Society that it was not invented for the treatment of sunken noses, and that Continental surgeons have used it, with good success, over a very wide range of cases, especially for the cure of prolapse of the rectum or of the vagina, with loss of control over fæces or urine. But my experience has been limited to nose cases; and of these I have had twenty-five, because my name, through no merit of my own, and no fault, got into the papers. I have taken every case as it came, except one that was too trivial for treatment; six were in private and nineteen in hospital practice; eight were female and seventeen male; the youngest was 19 and the oldest was 52.

In two cases the nose was not only somewhat sunken, but was also crooked, and had to be straightened with Walsham's forceps. In several cases there was perforation or destruction of the cartilaginous septum; in one there was also extensive destruction of the roof of the mouth; in one there was ozæna. Of the whole number of cases, about half were due to congenital deformity or to disease, and about half to injury; and the span of time between the injury and the treatment ranged from a few months to half a century. In a few cases some operation had been done to remedy the deformity, and had failed. The deformity ranged from a mere falling-in of the soft tissues below the bridge to the absolute flattening of the nasal bones, leaving hardly more than the tip of the nose upstanding from the face.

In my earlier cases I used Eckstein's paraffin, which melts at the very high temperature of 136° F. It was said to be safer and more permanent, and free from the very least risk of causing embolism; it was the latest modification, and I supposed it must be the latest improvement. So I used it in more than twelve cases, and I am quite certain now that it has no sort or kind of advantage over a paraffin melting

twenty-five or thirty degrees lower. It has, on the other hand, obvious disadvantages; it is very hard to work with, it sets with most embarrassing rapidity, and it is so hot that one runs the risk of burning the tissues. I have not a shadow of doubt that Eckstein's paraffin is a step in the wrong direction; but I am unwilling to go so far the other way as to use Gersuny's paraffin, which melts at 104° F.,—partly because there is some reason for thinking that it would yield a little if the patient had, for example, malaria, with a frequent temperature of 105° but more because the skin in these nose cases exercises immediate and very heavy pressure on the paraffin, and one wants a paraffin that shall set quick, and rigid, to stand this tremendous strain. Somewhere between 108° and 115° is, I think, the right melting-point; I am using at present a paraffin that melts at 111°, and it seems to me to have every advantage and no defect.

Whatever we use, we have to remember that when it sets it shrinks; and the paraffin that sets hardest shrinks most. This shrinkage is inevitable. Happily it is very slight; it may be only just visible; but there it is, and, in almost every case, you are a little bit disappointed when you next see the patient. The only thing to do is to add a few drops more of paraffin about a week later; no anæsthetic is needed except cocaine; and this necessity of "touching up" the nose is no argument against the value of the treatment.

With regard to syringes, I have tried several. At present I am using an Eckstein's syringe, but with a short, straight, steel needle. I think it would be hard to improve on this instrument; it keeps its heat perfectly, it is easily worked with one hand, and it has a screw-nut which prevents all possibility of a sudden rush of paraffin.

Everything should be prepared as for a major operation, and a general anæsthetic should be given. The melted paraffin and the syringes are put ready in a water-bath kept seven or eight degrees above the melting-point. The skin of the nose is just nicked, that the needle may be used lightly and with accuracy, but it is not a good plan to try to free or raise the skin beforehand with a tenotomy knife; only, if there has been exfoliation of bone, leaving a depressed cicatrix, it may be advisable to free the skin a little at this one point.

In one case, where I did try to free the skin somewhat widely, the mucous membrane also was punctured, and there was bleeding. I am inclined, therefore, not to meddle with

the skin; and the paraffin will raise it well enough without the help of a tenotomy knife.

Five or six cubic centimetres of paraffin are drawn up into the syringe, and it is held in the bath while the screw-nut is adjusted. Then it is taken out, and the needle is just dipped for two or three seconds in boiling water. It is then put well under the skin, below the level of the bridge and a little to one side of the middle line, and directed upward. The assistant keeps up firm pressure, making his fingers and thumbs meet in a ring all round the area of injection, taking great care to keep the paraffin off the forehead and the orbits. The injection is made about the rate of 1 c.c. every ten seconds. It is easier said than done, to get in all the paraffin through a single puncture; and, except in the most favourable cases, two or even three punctures are necessary. The moulding must be done vigorously; the paraffin must be squeezed hard and steadily till it seems absolutely rigid. The punctures are sealed with collodion, a mask of wet lint is laid over the nose, and the patient, so soon as he comes out of his anæsthetic, is given a hand-glass and is told to look after himself, and to mould his nose gently now and again during the rest of the day. Till he is well out of his anæsthetic he must be carefully watched; for in one case just lately the patient suddenly rolled over on to his face, and a small portion of the paraffin was pressed upward, making a thickening over the glabellum. In a few cases I applied a Walsham's external nasal splint, but I gave this up when I realised that the slight settling-down of the paraffin, just after the injection, is due to its natural shrinkage and cannot in any way be avoided.

With regard to the results in these twenty-five cases, I have had no disaster of any kind—no embolism, sloughing, or any other calamity of surgery. After a fairly long hunt through British, American, German, and French journals, I can find only one case of embolism; here it is probable that a vein was pierced—the injection was followed by "amaurosis," —but I cannot get at the original reference to this case. Embolism, in two cases at least, has followed the injection of paraffin into the loose and vascular submucous tissue of a prolapse; but I cannot find any evidence, except the one case which I have mentioned, that these nose cases are in any danger of embolism.

In one of my cases, about ten days after the injection, a small abscess formed and broke just below the inner angle of

the orbit. In two cases the line of puncture of the needle broke down. In a few cases some redness of the skin followed the injection, and in one or two of these, though it is much improved, it has not yet altogether gone. I attribute these defects of treatment partly to my own fault, but more to the use of Eckstein's paraffin. I have no other confessions to make, except that I have failed more than once to mould the paraffin with complete accuracy, so as to be satisfied with my own work. Indeed, with these nose cases it is impossible ever to feel perfect pride in the result; they are none of them above criticism; a faultless Greek nose, like a poet, *nascitur, non fit.* In some cases where the face is short and small and square, there is no room between the forehead and the upper lip for a Greek nose; and, if there were, such a nose would be out of proportion to such a face. In some cases the nose is so shrunk by syphilitic necrosis that it is contracted upward, and the upper lip is elongated; you cannot confer good looks on such a patient. In other cases the lower part of the nose is coarse and heavy, with large, ill-shaped nostrils and thickened skin; here, again, you cannot hope for complete success.

But, short of perfection, a vast amount of good can be done. I think that the worst cases give the best results; you may fail to straighten perfectly a nose that is only a little sunken, and you will succeed in giving size and shape to a nose that has been absolutely flat and ape-like for twenty or thirty years. You can be sure of raising the skin between the eyes, so that you fashion a fairly good-looking upper part of the nose; and by doing this you may change the whole character of the face, making it look more intelligent and less animal, and improving the look of the eyes. You can slightly increase the length of the nose, both in its real length and in its apparent length; and you can more or less depress the tip of the nose, so that the nostrils look more directly downward. Finally, it is worth noting that two of my patients, without being questioned, declared that they breathed much more freely through their noses after the injection than before it. At present I despair of making perfect noses; but it is easy, in nine cases out of ten, to improve a badly sunken nose so that it shall be at least unnoticeable, and shall not prevent its owner from getting employment, or from getting married, and shall not compel him to be laughed at, or pitied, or suspected of syphilis, wherever he goes. I say, in nine cases out of ten; for, of course, no two cases are alike. To-day,

for example, I have had two cases. One was a woman of 52, whose nose had been absolutely flat ever since she was 2; the other was a girl of 20, whose nose had been flat ever since she was born. The two cases appeared very much alike, but in the case of the older woman I have failed to do much good; in the case of the girl it was perfectly easy to raise the skin and to make a very fairly good-looking nose.

Of course one must have good assistance, and for this I am very grateful to Mr. Aubrey Pollock, my house surgeon, and to Dr. Shuter, anæsthetist to the West London Hospital.

XII.—*A case of Generalised Miliary Tuberculosis associated with Acute Synovitis.* By H. D. Rolleston, M.D. *Read January* 9, 1903.

A MAN æt. 52 years was admitted under my care in St. George's Hospital on November 14, and died on November 19, 1902. On admission the temperature was 103°, pulse 132, and there was pain and swelling in the wrists, knees, and ankles. He was semi-delirious, deaf, and slightly cyanosed. The present illness began two weeks previously with headache and pain in the back. He said that three years ago he had a similar illness lasting ten weeks. He had been a sailor, and had often had malaria. He was given salicylates freely on admission, and after this the pain seemed to diminish. Next morning his pulse was 106, and the temperature touched normal, but rose the same evening and remained raised (between 101° and 105°) until his death. The tentative diagnosis of acute rheumatism, made on admission, was subsequently given up, as the patient's general condition suggested an acute hæmic infection such as pyæmia, generalised tuberculosis, or malaria. No physical signs of any importance were made out; the blood did not show any malarial parasites; the retinæ were examined, and though the patient's restlessness rendered this difficult no optic neuritis or tubercles in the choroid were made out. The urine contained a trace of albumen, and did not show Ehrlich's diazo reaction. The patient got more prostrate, his mental condition worse, and his motions and urine were passed under him. The joints continued to be swollen and painful, and there was general tenderness all over the body. There was very profuse perspiration, and some twitching of the right side of the body before death, which took place late on November 19.

The diagnosis lay between severe typhoid fever, pyæmia, and generalised tuberculosis; the patient's condition resembled the latter, but the presence of distinct synovitis, which I had never heard of in generalised tuberculosis, made it appear more probable that there was some other form of hæmic infection.

At the careful and complete autopsy performed by Dr. R. S. Trevor forty-two hours after death, the left knee-joint contained an excess of synovial fluid, but there was no pus; the synovial membrane showed a number of petechial spots. Unfortunately no bacteriological examination was made. The mitral valve showed old endocarditis, but otherwise nothing remarkable was found in the body. Miliary tubercles, though carefully looked for, were not detected, and no tubercle was found in any part of the body. Pieces of the lung and spleen, however, when hardened for microscopic examination, were manifestly riddled with miliary tubercles. This was perfectly evident to the naked eye, and was fully confirmed by the microscopic examination. No primary tuberculous focus was found.

Remarks.—Clinically, the association of acute synovitis with generalised tuberculosis must be extremely rare, as I have not found any mention of it in the works of reference I have consulted. It is quite conceivable—at any rate the possibility cannot be excluded in this case—that there was another blood infection in addition to generalised tuberculosis, and that the synovitis was thus produced. But the fact remains that acute swelling of the joints may be a prominent feature during life in a case which after death shows generalised tuberculosis. An erroneous diagnosis of acute rheumatism may thus be made.

Another clinical point to which attention may be directed is the presence of general tenderness. I have seen this before in generalised tuberculosis, and am inclined to believe that it is due to wide-spread miliary tuberculosis of the muscles and tissues, and analogous to the tenderness of trichinosis. Possibly wide-spread tenderness is by no means rare in generalised tuberculosis. The fact that the miliary tubercles, although anxiously looked for in order to verify a tentative diagnosis, were missed at the autopsy, is of interest. Some years ago I examined a case of pneumonia after death, and only found out by microscopic examination of the lung that there was miliary tuberculosis as well as pneumonic consolidation.

XIII.—*A case of Hæmorrhagic Typhoid Fever.* By E. W. Goodall, M.D. *Read January* 9, 1903.

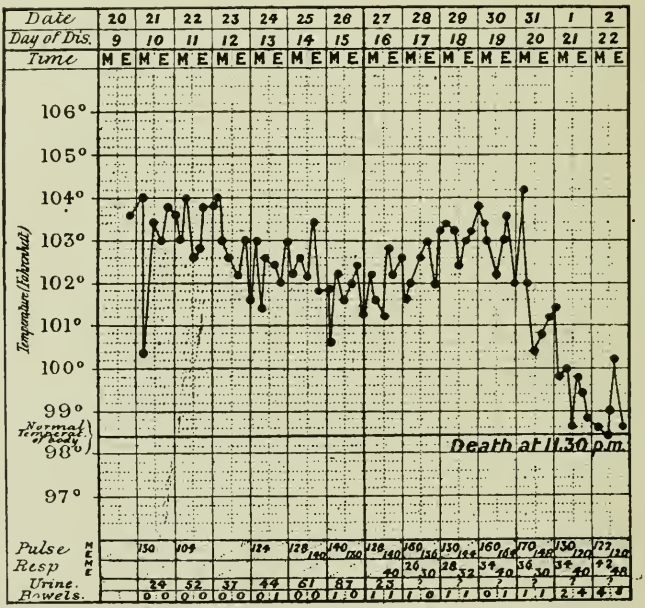
THE patient in this case was Mary C., æt. 26, an assistant nurse at the Eastern Fever Hospital, Homerton. She entered the service of the hospital in November, 1898; since that date she suffered at different times from scarlet fever, diphtheria, and rubella. At the commencement of her fatal illness she was employed on night duty. She made no complaint till the evening of January 20, 1902, when she said she felt too unwell to get up and go on duty. She was seen by one of the assistant medical officers, who found her temperature raised, some purpuric spots on the legs, and an erythematous rash diffused over the trunk, which led him to suspect scarlet fever. The young woman was complaining of headache; she stated that she first noticed the spots on the legs when she was going to bed that morning, and that she had been suffering from headache and pains in the limbs and back since January 12. There was no shivering and no vomiting.

January 21.—At 2 A.M. the patient's temperature was 104° F.; pulse 130, compressible; heart normal; lungs normal; skin yellowish, but not bile-stained; expression of face anxious; mind quite clear. On the front of the legs below the knees were a number of purpuric spots; there were also similar spots on the upper parts of the chest and arms, the lower part of the neck, and about the axillæ. Most of these spots were about the size of a large pin's head, though some of those on the shins were petechial. In addition to these cutaneous hæmorrhages was one blue subcutaneous hæmorrhage on the lower and outer part of the right upper arm. Blood was oozing from the gums and lips, and there were hæmorrhages in the mucous membrane of the mouth. Although it was not due for a fortnight, menstruation had commenced. The urine was of natural appearance. The erythema disappeared before morning. (For further records of the temperature and pulse-rate see the chart which is appended.)

January 22.—Some more purpuric spots have appeared on the arms, and there is a subconjunctival hæmorrhage on the right side. The menses are more copious, and contain more

blood than is usual for the patient. At 2 P.M. a pint of opaque urine, of the colour of burnt sugar, was passed. It gave the reactions for blood. Yesterday morning the patient was put on a mixture containing calcium chloride, 15 grains every four hours; this evening 10 minims of a 1 in 1000 solution of adrenalin chloride was ordered instead, every four hours.

January 23.—There is a large fresh hæmorrhage into the buccal mucosa on the right side, and some more blue subcutaneous hæmorrhages on the right hand and forearm. There is less bleeding from the gums and much less men-



strual flow; but the condition of the urine is about the same. Pulse small; patient put on champagne. In the afternoon she was prostrate; she was anxious, and as she was also unable to sleep she was given a $\frac{1}{2}$-gr. opium pill, which had the desired effect.

January 24.—Has vomited two or three times since last night; vomit "coffee-grounds" in character. Very little oozing of blood from mouth, and very slight menstrual flow. Breathing shallow and quick, but no abnormal signs in the

chest. Bowels confined; glycerine enema ordered. The urine is the same as before; when examined microscopically it was found to contain nothing but blood-cells.

January 25.—Pulse inclined to be running; retching at times; some bleeding from the mouth during the night; still a little hæmorrhage from the uterus; the urine contains blood; skin has a yellowish tinge. Since the commencement not many more cutaneous hæmorrhages have appeared; they are mostly on the limbs; the large subcutaneous hæmorrhages are on the thighs, upper arms, and forearms, and there is one on the left hand. There have been no violet or ink-like spots or effusions of blood, as in variola. The breath is very fœtid, and blood collects in a crust on the lower lip. The bowels were moved after the enema yesterday; the motions were very black and streaked with bright blood. Up to to-day the patient has been taking food pretty well, but to-day she is not.

January 26.—No bleeding from the mouth or uterus to-day. Patient looks very ill, and is worse. Vomiting during the day. The urine, which was clear and almost transparent all day, became turbid and brown in the evening, and deposited a thick, chocolate sediment. The patient is becoming restless, but is at the same time languid and disinclined to speak; at times she is drowsy.

January 27.—Retching occasionally; urine a little darker than yesterday; mind quite clear; a little oozing of blood from the month.

It is hardly necessary to give details of the case between this date and the patient's death, which took place at 11.30 P.M. on February 2. No fresh hæmorrhages appeared, and as far as appearance went the urine became free from blood. The pulse and respiration rate were high; the breathing became shallow and there was an occasional cough; till two days before death the temperature was considerably raised. Up to January 27 the bowels had been confined; after that they were opened once or twice daily, the motions being formed. On February 1 and 2 they were opened more often, the stools being small, clay-coloured, but not very loose; at no time were they "pea-soupy." The patient was restless, had only snatches of sleep, and wandered at times; towards the end she became unconscious. On January 30 she had lost the ghastly, yellow look she had previously presented, and had become extremely pallid. There was occasional retching. The pulse very gradually

failed; indeed, for the last five or six days her death was hourly expected. During the last few days there was incontinence of urine and fæces.

A *post-mortem* examination was made on February 3 at 5.30 P.M. Rigor mortis present; universal yellowness of skin, not due to bile; body in a good state of nourishment; fat more yellow than usual. The purpuric spots observed during life were still present, though somewhat faded. They were to be seen on the face and chin (a few only); lower part of neck and upper part of chest; upper extremities, especially on the extensor surfaces; upper and outer parts of thighs; shins and dorsa of feet. On the shins and feet the spots were petechial and of a very red colour; elsewhere they were larger and of a faded purple hue, two or three lines to a quarter of an inch across. On the arms, hands, and thighs were also a few larger subcutaneous hæmorrhages, half an inch to one inch in diameter. There was a small subconjunctival hæmorrhage in the left eye.

Many hæmorrhages were found in the parietal peritoneum. There was also a good deal of hæmorrhage in the connective tissue round the right kidney, and some into the omentum and mesentery. No peritonitis.

Spleen, 4½ oz.; quite firm and normal. *Kidneys:* left, 5½ oz.; pale and somewhat blurred, otherwise normal; right, 8½ oz.; round it much extravasation of blood; pelvis and calices full of recent blood-clot; many small hæmorrhages into substance, especially near the surface; many groups of yellowish spots, size of sago grains, seen on surface; they stood out above the surface; when cut into they showed the same appearance and extended deeply into the organ; structure of kidney coarse and mottled. *Adrenal bodies*, normal. *Pancreas*, normal. *Liver*, pale, but normal. *Gall-bladder*, small, normal; contained yellow mucus. *Stomach*, pyloric end showed a very large number of petechial hæmorrhages over an area of several square inches. *Uterus*, normal. *Ovaries:* right, a large Graafian follicle full of recent clot, half an inch across; left, a similar follicle, but older, with breaking-down clot. *Bladder*, many submucous hæmorrhages, some of them half an inch or more across.

The *intestines* showed many large submucous hæmorrhages, especially in dependent loops. A good many ulcers were found, the highest 2½ feet above the ileo-cæcal valve. These ulcers were situated in Peyer's patches and solitary follicles, mostly the former, except near the valve, where for three or

four inches was a good deal of irregular ulceration; except here the ulcers were mostly round. Their edges were clean-cut and slightly undermined; there were no sloughs anywhere. All except three or four of the smallest ulcers were situated in the midst of patches of submucous hæmorrhage, so that the question was raised whether the ulcers were not the result of the hæmorrhages. But I thought not; they were too defined and deep, and they looked just like typhoid ulcers of the third or fourth week. There were a good many small round ulcers in the upper half of the large intestine. The mesenteric glands were considerably enlarged and infiltrated; into most of them hæmorrhage had taken place.

Heart, normal. *Lungs*, old adhesions over nearly the whole of the left lung and between the lobes of the right. The posterior and lower parts of both lungs were extremely œdematous and airless, but there was no consolidation or recent pleurisy. No other organs were examined.

A *microscopical examination* of the various organs was made by Mr. Dunstan Brewer, who has kindly furnished the following description:

Spleen, normal; no organisms to be seen in the sections made. *Kidneys:* left, cloudy swelling, with slight desquamative nephritis; no organisms; no hæmorrhages; right, cloudy swelling and focal necrosis; early desquamative nephritis and glomerulitis. The small white patches are septic foci, consisting of masses of small round-cells showing little tendency to necrosis. In typical masses the centre consists of a mass of organisms (bacilli, ? *B. coli*). The tubules are filled with *débris*, chiefly consisting of round-cells and broken-down blood. There are numerous hæmorrhages, mostly recent. Both the hæmorrhages and the septic foci are mainly in the medulla. There are no cocci in either kidney.

Pancreas, normal. *Liver*, slight granular degeneration, with small masses of young connective tissue and early necrosis in parts; no fat; no hæmorrhages; plugs of streptococci in capillaries. *Uterus*, normal. *Heart*, slight granular degeneration; no vacuolation; increase of intermuscular tissue; small intermuscular hæmorrhages; a few masses of streptococci in vessels. *Lungs*, patches of broncho-pneumonia, in places breaking down into pus; general œdema; masses of streptococci, chiefly in and near vessels and suppurative foci.

As no Widal's test had been done during life, some of the blood was taken from the heart and the test tried *post mortem*. The reaction was observed well within half an hour

with a 1 in 20 dilution, but only very slightly within half an hour with a 1 in 40.

Agar cultivations were taken at the autopsy from the heart-blood, the spleen, and a mesenteric gland. Dr. Cartwright Wood very kindly examined the resulting growths. The culture from the heart-blood gave a growth of *Bacillus coli*, while those from the spleen and mesenteric gland resulted in growths of *B. coli* and *B. typhosus*. The latter gave all the reactions, including agglutination with a specific serum. Organisms that liquefied gelatine were also obtained from the mesenteric gland, but they were not further identified.

Clinically this case was very obscure. The nurse had not been employed in a typhoid fever ward. At first the symptoms were only those of acute purpura hæmorrhagica. The patient had been vaccinated in infancy, and again on entering the service of the hospital in 1898, and had seven very good marks on the left arm; so that the facts as to vaccination and re-vaccination, quite apart from the absence of other symptoms, negatived the idea of smallpox, which was very prevalent at the time. After a day or two I thought that the hæmorrhage was only symptomatic of an acute general infection (? streptococcal infection or something of that nature). On the evening of January 29 I began to have suspicions of typhoid fever, but I never had more than suspicions. I examined the patient carefully on that night, as well as on other occasions, but could find no definite evidence of typhoid fever; but I noticed that the abdomen was a little full and the muscles a trifle resistent. No spots were ever seen, either by myself or by Dr. Basan, under whose immediate care the patient was. Nor was there anything in the condition of the bowels to suggest typhoid. Yet the *post-mortem* examination showed that the case was certainly one of typhoid fever.

It is a well-recognised fact that hæmorrhage into the skin and organs and from mucous surfaces may occur with varying frequency in several of the acute infectious diseases. In smallpox and diphtheria, indeed, this complication is common; but in typhoid fever it is very rare, and it is partly on this account that I publish this case. Trousseau mentions the condition under the name of "hæmorrhagic putrid fever," but the case he relates was not a marked one. Murchison simply mentions the fact that such a form of the disease is sometimes met with. Curschmann, the writer of the article on typhoid fever in *Nothnagel's Cyclopædia*, states that he has observed only six cases; he quotes Liebermeister as

having seen three among 1900 cases of typhoid fever in an epidemic at Basle, and Weil one in 150. Osler, the editor of the English edition of Curschmann's article, adds that at the Johns Hopkins Hospital only one case occurred amongst 829 cases of typhoid fever.

On looking up the notes of the cases at the Eastern Hospital, Homerton, I find that the case I have just related is the only marked one occurring during the period I have been connected with the hospital, from January, 1892, to December, 1902—eleven years. During this time the number of typhoid fever admissions has been 1733, including cases occurring amongst the staff. So that the complication is decidedly rare. I find, however, that there have been fifteen cases, including the present one, in which hæmorrhage other than intestinal has taken place. In fourteen of these cases there was hæmorrhage, either purpuric or petechial, often both, into the skin; and in one from the gums, lips, and palate, but not into the skin. Amongst the fourteen cases in which it occurred in the skin it occurred in other places as follows:—Fauces, one case; nose, one case; pleuræ, one case; lungs, one case; nose and mouth, two cases (one of these cases had cancrum oris at the same time); beneath the conjunctiva and into the mesentery, pleuræ, and larynx, one case; nose, mouth, and uterus, one case; and lastly, the present case, in which the eye, mouth, uterus, kidney, peritoneum, bladder, and stomach were affected. There were, therefore, five cases only in which the skin alone was involved; one of these cases was fatal from pneumothorax, but the patient was seriously ill with typhoid fever before this complication arose.

All but three of the fifteen cases died; in one of the cases that recovered the skin alone was involved; in another there was bleeding from the fauces; and in the third from the nose. In none of these cases were the hæmorrhages extensive or severe. An extensive purpuric or petechial eruption, therefore, in typhoid fever justifies a very grave prognosis.

Nine of the fifteen patients were between the ages of five and fifteen years; the oldest was a man of thirty, the youngest a girl of five. Eight of the patients were males and seven were females.

In none of the cases except the present had the hæmorrhagic condition arisen earlier than the second week of the disease; this case is unusual in that hæmorrhages were amongst the first noticeable indications of illness.

The etiology of hæmorrhagic typhoid fever has been well

the point of the forefinger is with difficulty passed down to the acetabular surface. The head of the femur, before the age of three, is, in my opinion, normal. It then becomes flattened at one part. The flattening gradually extends over the whole surface, and thus the head of the bone becomes deformed, and as the result of subsequent osteo-arthritic change, ultimately disappears. The ligamentum teres I have considered hitherto to be absent, having only once felt the remains of one during operation; but it is quite possible that I may have overlooked a thin flaccid band such as exists in the specimen which I have submitted.

In many cases the neck of the femur is curved, and this is specially the case on one side in cases of double dislocation. The curve presents usually a concavity anteriorly, so that when the patella is directed forwards the head of the femur looks forwards as well as inwards, instead of directly inwards. Various groups of muscles are more or less contracted in this condition. Thus the adductors are always considerably shortened, the tensor vaginæ femoris, sartorius, and the front part of the gluteus minimus and medius are nearly always somewhat contracted, and the hamstrings occasionally. The iliacus and psoas tendon, in consequence of displacement of the femur upwards and outwards, passes round the anterior inferior spine, outwards and backwards, and, in some cases, even upwards, to the small trochanter; thus, in severe cases, the pelvis is partly suspended upon the psoas tendon. The muscles of the leg generally on the affected side are somewhat wasted, and the pelvis is apt to be flattened and oblique upon the affected side; and lastly, in consequence of the dropping of one side of the pelvis in unilateral cases, scoliosis is subsequently developed. In bilateral cases lumbar lordosis exists from the first.

As regards symptoms, suffice it to say that only occasionally has the mother noticed a flattening of one buttock prior to the child's walking. These children do not walk till late (fourteen to twenty-four months), and then in unilateral cases with a limp, or in bilateral with a waddling gait. The shortening slowly increases from that time until about puberty, when it undergoes a rapid increase, and subsequently there may be a slight diminution in the shortening. The shortening is greatest, possibly four inches, about the age of twenty.

I am unable to offer a definite opinion as regards etiology. I do not believe it is due to a defect in ossification of the

floor of the acetabulum. The condition is developed long before the Y-shaped cartilage becomes ossified. The tendency to this condition appears in some cases to be hereditary: in two of my cases, aunt and niece; in two others, mother and daughter; and in five other cases, three in one and two in a second family.

I have met with it associated with genu recurvatum in three cases, and at one time I was inclined to believe that congenital dislocation of the hip was produced as the result of malposition *in utero* leading to spontaneous subluxation and maldevelopment of the acetabulum subsequent thereto. It is possible that it is the tendency to the deficiency of liquor amnii which is hereditary, and hence the apparent heredity of the dislocation.

It is difficult to explain the simultaneous occurrence of other congenital deformities, such as cleft abdomen, as being due to deficiency of liquor amnii.

The treatment of this condition by ordinary orthopædic methods—namely, recumbency and continuous extension by weights with or without tenotomy, followed by extension instruments—is unsatisfactory. This treatment necessitates the wearing of an instrument for very many years, and the result is simply to decrease the amount of shortening by one inch or so; and as soon as the instrument is left off the shortening again increases. Neither is the method practised by Mr. Reeves, of manipulating the bone so that the head becomes placed underneath the anterior inferior spine and is then fixed by plaster, attended with very satisfactory results. In my opinion, treatment in these cases resolves itself into one of two methods: either that recommended by Paci and subsequently developed by Lorenz—namely, after tenotomy and extension, manipulation of the femur so that the head of the bone is brought to rest upon the acetabulum,—or secondly, to deepen the old acetabulum as practised first by Hoffa, but through the incision recommended by Lorenz.

With regard to the first of these two methods, I think the specimen I have shown this evening will convincingly demonstrate that it is impossible to so manipulate the head as to place it within the acetabular cavity. All that can be done is, by manipulation, to pass the head over the posterior rim of the acetabulum and make it rest upon the constricted part of the capsule which covers the acetabulum. It will readily be understood, I think, why congenital dislocation of the hip treated by this method is so prone to re-dislocation.

Of some twenty cases which I have treated by this method during the past six years, only three may be said to be very good results. The rest of the cases, which have not been lost sight of, already present reluxation, more or less severe.

I have been led, therefore, to add the open operation, after reposition has been effected by tenotomy and manipulation. The following is the method I adopt, in a given case:

After a stirrup of strapping has been applied to the leg and the usual preparations made, the patient is anæsthetised, the leg forcibly extended by blocks and pulleys fixed to the wall of the theatre, counter-extension being provided by means of a long roller towel, which, passing over the pubes, protected with cotton wool, projects above the shoulder of the patient on the same side as the dislocation. The leg is then forcibly stretched for five or ten minutes. Care must have been bestowed upon the application of the plaster stirrup; otherwise it is dragged off the leg. This stretching is painful, and must be judiciously applied, or the subsequent shock is considerable. The apparatus is now removed and an assistant fixes the opposite iliac crest. The affected thigh is flexed, rotated inwards, and forcibly abducted upon the operator's fist, placed under the great trochanter as a fulcrum.

In young children under the age of four it is possible to lift the head of the femur forwards over the posterior rim of the acetabulum, after which the leg is extended, being still in the abducted position. Should the patient be older than three or four years reposition is very rarely possible at the first attempt. In these cases free subcutaneous tenotomy of the adductors longus, medius, and gracilis, and tensor vaginæ femoris and other tight muscles, *e. g.* sartorius glutei, hamstrings, must be performed. The patient is dressed and sent to bed, an extension weight of 6 or 8 lbs. being applied. A fortnight later the stretching is repeated, and after massage of the divided parts a second attempt is made to replace the head. It may be necessary to repeat this procedure three, four, five, or even six times, each at intervals of a fortnight. If the child is below the age of ten, one, two, or three sittings may suffice; but the older the patient the more is the shortening and the greater the difficulty in getting the head over the posterior rim of the acetabulum by manipulation. After the age of twelve this is well-nigh impossible, and the oldest patient in whom I have succeeded in reducing the dislocation after tenotomy and repeated extension was a girl of fifteen.

After repeated attempts I failed to replace the head of the femur in one girl aged fourteen. Considerable strength is occasionally necessary in order to effect this reposition. Fracture of the neck of the femur, therefore, and separation of the epiphysis of the femur, are accidents which occur and must be borne in mind when manipulating these joints. Such an accident necessitates, at the very least, the application of a long splint with extension for six weeks and the cessation of the treatment for a year. If reposition is to be the only treatment adopted, the limb must be put up in plaster of Paris in a position of least abduction compatible with the continuance of the head in the desired position. This position is often one of extreme abduction, the thigh making an angle of nearly 90° with the trunk. The plaster is allowed to remain on for a month or six weeks. It is then removed, the limb examined, the femur brought down into a position of less abduction, and the plaster reapplied for six weeks longer. It may take four or five sittings before the femur can be brought into the position of normal extension. I have, in my cases, reapplied the plaster at intervals, in some cases for as long as two years; and notwithstanding this long fixation of the limb, re-dislocation has subsequently occurred. I therefore feel that, except for young children under the age of four, this method of treatment is not satisfactory, and during the last three years I have supplemented this method by the open operation.

After the shortening has been overcome sufficiently to enable me to manipulate the head into the desired position, the patient is sent back to bed and extension reapplied as before. A fortnight later, under strict aseptic precautions, an incision is made from the anterior superior spine downwards along the anterior edge of the great trochanter, and at the centre of this a perpendicular cut is made backwards into the tensor vaginæ femoris and the anterior borders of the glutei. The front part of the capsule of the joint is reached without difficulty and is opened freely. On exploring the inner part of the distended capsule a small round opening is felt, and is slit open at several points with a curved blunt bistoury, the leg being manipulated, generally adducted and rotated out, by an assistant, so as to prevent the head of the bone interfering with this procedure. With this curved scoop—which was made for me by Messrs. Krohne and Sesemann in 1894, and which fits the hand and forefinger—I remove the glenoid ligament and

sufficient of the cartilage to form a deep wide acetabulum. The joint is thoroughly irrigated, so as to remove all fragments which may have been left behind. Finally a small portion of the cartilage of the head of the femur is removed. The bone is next manipulated into the new acetabulum. A drainage-tube is inserted, the wound sutured and dressed by iodoform, aseptic sponge, carbolic gauze, wool, and bandaged. By the sixth day the drainage-tube is removed and the wound re-dressed.

Of the six cases which I have operated upon during the past two years, five healed up by first intention and all six are well. In no case has there been ankylosis. In no case, as yet, has there been re-dislocation. From month to month the condition of the joint improves. In one case which I operated upon seven years ago, a girl has three quarters of an inch shortening, and may be said to walk almost as well with one leg as the other.

Discredit was thrown upon this operation some years ago by a case which was said to have been cured in Germany with a movable joint, but where, subsequently, firm ankylosis of the hip was demonstrated—I believe to this Society. My experience is that if the cartilage of the head of the femur is not removed, no ankylosis develops. My object in removing a *small* portion of the cartilage is purposely to get an adhesion which may replace the ligamentum teres. I usually keep the limb in plaster of Paris for eighteen months or so, and in no case has ankylosis resulted. If any open operation is undertaken, in my opinion it should be that of deepening the old acetabulum. All other procedures—such as subcutaneous scarifications of the interior of the joint, as recommended by Brodhurst; the removal of the head of the femur, with or without fixation of the femur to the ilium; the manufacture of a new acetabulum above and behind the old one; the detachment by chisel and the drawing downwards of an osseous and periosteal flap to the ilium, as recommended by König; the injection of chloride of zinc round the joint, as recommended by Lannelongue; the injection of iodine tincture into the joint, or the removal of a portion of the capsule itself—are attended with equal danger to the patient and effect no useful object. I feel confident that if the shortening is overcome beforehand by repeated extension and tenotomy, so that the head of the bone can at once be manipulated into its place so soon as the acetabulum has been deepened, the shock from this operation will always be found to be slight,

and there is no reason, in my opinion, why it should ever be attended with a fatal result. It is by this procedure only that a new joint perfect in all respects as regards equilibrium and motion is possible. After this operation the tendency to re-dislocation is reduced to a minimum, and with careful treatment may be avoided. Moreover the hip-joint daily becomes more mobile and more useful.

XV.—*Cases illustrating Acute Suppurative Arthritis of the Knee-joint.* By H. L. Barnard. *Read February* 13, 1903.

An acute pyogenic infection of the knee-joint is one of the most dangerous of suppurative diseases, and one of the most unsatisfactory class of cases that a surgeon can be called upon to treat.

Other collections of pus in the body can be efficiently and reliably drained, and are attacked by the surgeon with confidence, but when the knee-joint is filled with pus a feeling of helplessness and futility appears to overshadow the treatment.

The results only too well justify this feeling. Many of these suppurative arthrites are produced in hospital, and follow wiring of patellæ or removal of semilunar cartilages or foreign bodies. They are under constant medical supervision, and therefore are recognised early. They receive prompt attention, and are afterwards treated with all the resources at the command of the surgeon who has had this unfortunate accident; yet what are the results? Many deaths, more amputations; and the rest, after months of suppuration, leave the hospital in broken health with an ankylosed knee and often open sinuses.

The method adopted to drain the knee-joint is usually to make lateral incisions two or three inches long on either side of the patella, and to pass tubes from these across the joint and up into the subcrureus bursa. The joint is then irrigated with some antiseptic solution, such as carbolic lotion 1 in 40, or perchloride of mercury 1 in 1000. The leg is then placed on a back splint and surrounded with copious fomentations or dry dressings.

Mr. Jacobson, in his *Operative Surgery*, when speaking of draining the knee-joint after wiring the patella, says that should it be decided to do so, "drainage should be employed through the wound to the most dependent part of the joint at the outer side (Lister), thrusting the instrument here through

the joint and soft parts, cutting upon it, and drawing a drain through."

It was an attempt and a failure to do this in a case of suppurative arthritis which induced me to carry out this investigation of the anatomy of the knee-joint, and led me to plan the posterior incisions recommended in this paper.

In 1888 Sir Frederick Treves advocated, in the *British Medical Journal* for July 7, continued irrigation in such cases with some weak antiseptic. By blocking the outlet tube the joint was distended with the fluid once or twice a day. He published two most hopeful cases in which good movement was obtained after the treatment.

Quite recently one of Sir Frederick Treves' cases came to my out-patients' at the London Hospital for some other matter. His knee had practically perfect movement. Unfortunately the treatment has not led to similar results in a series of cases under several surgeons at the London Hospital.

There have been seven cases in the last ten years treated by this method. Two died of pyæmia; four had fibrous ankylosis after many weeks in hospital. One case was twenty weeks in hospital and left with open sinuses. Only one case obtained movable joint.

The great difficulty in considering the value of any treatment is to be sure it was applied to really bad cases of the disease it cured, and I am afraid the very hopeful results of continued irrigation are further militated against by the fact that the first two cases reported appear to have been mild ones. The first had been opened for chronic synovitis and drained. After a week or so the knee became infected, apparently when the tubes were removed. They were at once replaced and the knee more thoroughly drained. Continued irrigation was established a few days later. This joint, which was being drained even before it was infected, can hardly be considered on a par with the cases of punctured wound, where for some days the pus is retained in the joint under high pressure, and no doubt with increasing virulence. The second case followed a fall without a wound, and the temperature was only 101° F.

Many years ago a case of suppurative arthritis of the knee-joint in a child was treated by Mr. F. C. Eve in the London Hospital, first with ordinary lateral drainage, and then with continued irrigation; but as both these measures failed to efficiently drain the joint, and the temperature continued to range at a high level, he laid open the knee-

joint by a curved incision across the front of it, and divided the patella and fully flexed the knee, so that all parts were fully exposed. The result was satisfactory.

Mr. Walter Whitehead has recently reported a similar case in the *British Medical Journal*. The knee was infected whilst a loose cartilage was being removed. Free drainage and irrigation were adopted but failed. The patient was delirious, his temperature 103°, and he was in a state of extreme exhaustion. His knee was then laid freely open by a transverse incision, the patella was sawn across, and the crucial ligaments divided. The joint was packed with gauze after it had been thoroughly cleaned. The patient rapidly convalesced, and in fifteen days the granulating surfaces of the joint were placed in an extended position. He made a good recovery with a stiff, but straight leg.

This method is no doubt efficient in all cases in which the pus has not burst through the capsule behind, and is already tracking up and down the leg. But it is heroic, and in every case in which it is applied all hope of a mobile joint is necessarily relinquished. The leg is, moreover, greatly damaged by the free division of the quadriceps insertion and other muscles sending expansions to the capsule of the joint. In short, it should be, as indeed it is, a forlorn hope of conservative surgery when amputation is refused, or when the surgeon still hopes against hope to save a limb for the condition of which he is responsible.

It was this gloomy experience of the general failure of the present methods for draining the knee-joint which induced me to investigate the anatomy of this serous cavity, in order to discover why it was so difficult to drain it, and whether incisions could not be planned which should make the retention of pus within the cavity of the joint impossible. This figure of the complicated synovial membrane of the knee-joint is taken from Testat's *Anatomy*.

The great subcrureus bursa is well known, and where the joint is drained it is never overlooked ; but this is not the case with the complicated pouches at the back of the joint.

The knee-joint is divided into two main pouches behind by a septum projecting forward from its posterior wall and formed in order from behind by the posterior ligament, the crucial ligaments, and the ligamentum mucosum (Fig. 3). Each of these pouches receives the posterior protuberant extremity of the corresponding condyle in extension. These pouches terminate above and behind the condyles in sacs,

which are beneath the corresponding head of the gastrocnemius (Fig. 2, *c*). The outer pouch also sends a synovial prolongation along the popliteus tendon (Fig. 2, *d*). The usual method of draining the knee-joint by a tube passed below the patella, and another passing up into the subcrureus bursa, leaves these two posterior pouches untouched. The leg in such cases is kept extended on a splint, which closes the way to the front of the joint by a coaptation of the bony surfaces.

When these pouches rupture from distension with pus

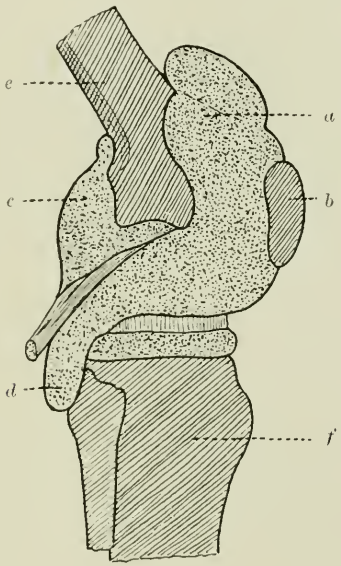


Fig. 2.—Lateral view of the knee-joint (after Testat), showing the anterior and posterior synovial pouches. *a*, subcrureus bursa; *b*, patella; *c*, the gastrocnemius pouch; *d*, popliteus pouch.

their anatomical position directs the pus up the thigh between the hamstrings and the great adductor, and down the calf between the gastrocnemius and the deep muscles.

It is difficult, if not impossible, to drain both these collections of pus at once. For if the leg be elevated the lower will drain, but the upper will pocket and spread further up the thigh, and *vice versâ* if the leg be lowered.

It is clear, then, that each of these posterior pouches must be drained by an incision placed as far back as possible. On the inner side, the knee being fully extended, the protuberant extremity of the condyle is cut down upon, and a pair of stout forceps are pushed under the head of the gastrocnemius and withdrawn open. A tube is then inserted. This is more easily done if the knee be flexed as the tube is put in, since this relaxes the head of the gastrocnemius and the posterior ligament. This incision will be found to be between the sartorius and gracilis tendons, and usually just behind the internal saphenous nerve.

In a similar way the outer condyle is cut upon. The incision here passes between the biceps tendon and the gastrocnemius, and just behind the external popliteal nerve. It is important, therefore, in both cases to be careful in the use of the knife, and to see that the incision is in the line of these nerves. When the skin has been incised it is easy to scrape down to the capsule with some blunt instrument. The knife can then once more be safely used to freely open the joint. Anatomical guides are useless in practice, because of the inflammatory swelling; one knows that the pouch surrounds the prominent condyle, which can easily be felt, and one cuts down until the cartilage of the condyle is reached. A drainage-tube should be passed across the front of the joint beneath the patella, and another directed up into the subcrureus bursa from either the inner or the outer side. This tube can with advantage be brought out through a median incision at the highest point of the joint anteriorly. The discharge from the anterior tubes rapidly diminishes, and the wounds heal some time before the two posterior incisions cease to discharge and close. This is proof in itself that the drainage is practically as well as theoretically correct.

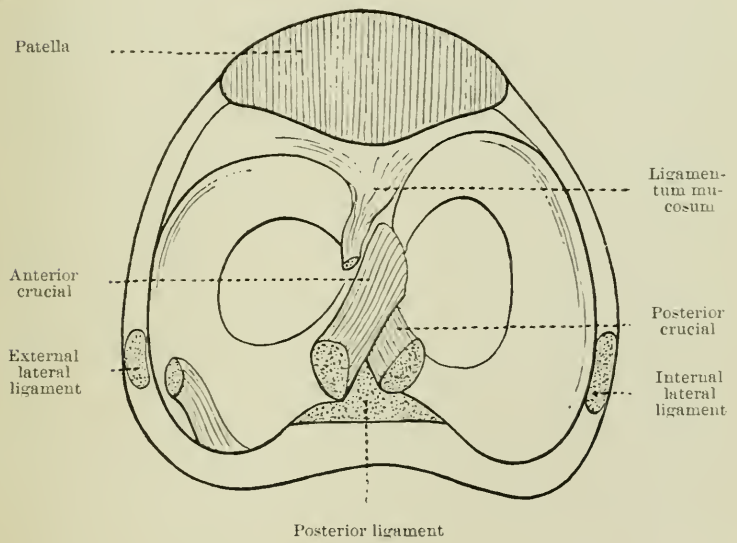
I have treated nine cases by this method, and in all the temperature reached normal in a day or two, appearing to show that no pus remained in the joint under tension.

The evacuation of the pus was greatly assisted in several cases by gentle stroking massage of the subcrureus bursa from above, and of the poplitens bursa from below up, movements which can be simultaneously carried out. As soon as the temperature is normal, I would recommend massage and passive movement. These must be carried out by a thoroughly efficient masseuse. The method is the same as for fractured patella, and is directed to similar ends. The patella is firmly grasped and moved about on the condyles of

the femur, so that it may not form adhesions to it. The knee is then steadily lifted from the bed so as to passively flex the knee-joint.

We have so far seen that anatomy indicates the presence of these large pouches behind the knee, and that they can easily and effectually be opened and drained. It only remains to narrate a typical case of acute suppuration I had an opportunity of seeing some time ago, in which this was not done, and these pouches ruptured and apparently involved the popliteal vein, leading to pyæmia.

Fig. 3.



An old and somewhat alcoholic man fractured his patella. After an interval of about a week the fragments were wired together. Unfortunately the knee became septic on the third day. The knee was opened by the usual anterior incisions and continued irrigation instituted. In about a week the patient died. At the autopsy, pus was found in the depths of the popliteal space tracking down into the calf around the vessels and up the back of the thigh. The lungs were studded with pyæmic abscesses.

Before narrating the nine cases I have treated in this way, I should like to say that they are consecutive.

The first four were traumatic cases, and have done much better than the last five, which were cases of auto-infection.

Case 1. *Wound of right knee-joint with rusty hat-pin; suppurative arthritis; drainage by anterior and posterior incisions; rapid recovery.*—This case was under the care of Mr. Mansell Moullin, who kindly gave me permission to use it. D. H., a little girl æt. 8, was admitted to the London Hospital on January 18, 1901. Seven days before admission she ran a rusty hat-pin into her right knee. The hat-pin was extracted by her medical attendant, and the wound healed. On admission the right knee was red, hot, swollen, and œdematous, and tightly distended with fluid which an exploring needle proved to be pus. Her temperature was 103° F., and her pulse 110. Her general condition was bad, and she suffered great pain from her knee. The same day Mr. Hugh Lett, who was house surgeon to the case, operated by the posterior incisions recommended in this paper, in addition to the usual anterior ones. Pus under high tension spurted from the incision on the inner condyle. The progress of the case was satisfactory. The temperature fell to 99·5° in three days, and reached normal in six days. The tubes were removed in fourteen days.

It is very interesting to note in this case that the anterior incisions closed some time before the posterior ones, a fact which seems to indicate that a posterior drain is right in practice as well as in theory.

This little girl was shown before the meeting this evening. She can run and walk well with her knee, which can be flexed nearly to a right angle.

Case 2. *Suppurative arthritis of the left knee-joint following lymphangitis and incisions; drainage by posterior incisions.*—Mark P., æt. 15, was admitted to the London Hospital on March 7, 1901. One month before admission he contracted a sore upon his foot from the rubbing of his boot. Lymphangitis spread up his leg and led to an abscess over the lower third of the tibia, and another over the internal condyle of the femur. These abscesses were opened by a general practitioner. On admission to the hospital the lad had a sinus in the lower third of his left leg, and another on the inner side of his knee, which entered the popliteal space. The knee was flexed somewhat and contracted. The temperature was normal. Two days after admission the house

surgeon straightened the knee under an anæsthetic, and thoroughly opened up and drained a large collection of pus lying in the depths of the space upon the knee-joint. Next day the knee became distended with fluid and very tender. The temperature rose to 103° F., and on the following day was 104° F. On the following day, March 11, 1901, I opened up the knee-joint by five incisions. Two were placed on either side of the patella, and a tube passed across the front of the knee-joint. One passed through the quadriceps into the upper extremity of the subcrureus pouch, and a tube was passed from this incision out of the inner lateral incision. Each condyle was cut on in turn from behind, and a tube inserted; as the joint was opened by the inner incisions pus gushed out. The temperature fell from 104° F. to 99° F. in thirty-six hours, and the leg became much less swollen and painful. It was some time before the sinuses closed, but the temperature did not again become raised above normal for twenty-four hours.

I have unfortunately not been able to trace this boy, and therefore cannot give his ultimate condition, which promised to be good.

Case 3. *Compound fracture of patella; suture; suppuration; drainage; recovery with good movement.*—This case has already been fully reported in the *Clinical Society's Transactions*. The chief points are these:—He was caught by an engine in August, 1901, whilst crossing the track, and struck upon his patella, so that this bone was crushed and the lower fragment projected from the wound.

The parts were freely exposed and thoroughly washed; the more ragged tissues were cut away; finally the fragments were brought together by a kangaroo tendon after Stimson's method.

For seven days the case did well; then cellulitis commenced, and in the course of the next two months much of the subcutaneous tissues round the knee sloughed away. The knee-joint became swollen and very tender. It was therefore opened on either side of the patella and behind, in the manner advocated in this paper. His knee was very slow to heal, but recovery was hastened by massage and passive movements.

The ultimate result was much better than could have been anticipated. The patella was united by bone, and the knee-joint could be flexed to a right angle. He could walk and run practically without a limp, and went back to full work.

It must be admitted, however, that this was a mild infection. As seen at the meeting to-night, the result is practically perfect as regards his knee, although he still appears to be suffering from traumatic neurasthenia.

Case 4. *Wound of the right knee-joint with glass; suppurative arthritis; drainage.*—A boy, Charles S., æt. 14, was admitted under my care into the London Hospital March 23, 1902. Ten days previously he fell on a piece of glass, wounding his right knee, which had been swollen ever since; but he was able to walk until two days before, when it became exceedingly painful and swollen. On admission, the knee presented all the signs of suppurative arthritis. His temperature was 103° to 104°. His general condition was good, but he had a tendency to be delirious at night.

March 23, 1902.—The knee-joint was laid open front and back, and drained with tubes.

Progress.—The drainage was not strikingly efficient in this case. The temperature slowly declined, but did not actually reach normal, and remained there for a month. The change for the better really occurred in his case when Mrs. Palmer, masseuse to the London Hospital, began to massage the joint and produce passive movement. She found that gentle stroking movements in front from above down towards the patella evacuated large quantities of pus, and that a similar result followed upward massage of the calf behind. Apparently the pus was bagging in these two situations, where, as we have seen, deep pouches of the knee-joint exist.

This case is shown to-night. The joint promised to have some movement when the patient left the hospital, but he fell soon after and wrenched it, and since then it has been quite stiff and flexed. I propose to take him into the hospital and straighten his knee, and give him a poroplastic splint to prevent re-contracture.

Case 5. *Suppurative arthritis of the left knee probably due to leucorrhœa; drainage; persistent sinuses.*—Emma F., æt. 23, was admitted to the London Hospital on July 18, 1902. Three days previously her left knee had become swollen and painful. Her temperature was 100° F. She was three months pregnant. No cause could be found for the suppuration except a leucorrhœal discharge. A needle was inserted into the knee-joint and pus withdrawn, which, although sent to the bacteriological department, was apparently lost. The

joint was then drained and irrigated with biniodide of mercury solution in a manner exactly similar to previous cases. For ten days after the operation the temperature remained absolutely normal. The tubes were then removed, and the temperature began to oscillate. The tubes were replaced, and several subsequent attempts were made to remove the tubes, but on each attempt the joint again became distended with pus, and they had to be replaced.

At a subsequent operation bare bone was found, extending some distance down the back of the tibia, and it seems probable that the mischief in the knee had originated in the head of the tibia.

When last seen the sinuses were still open. The temperature was normal, but the synovial membrane was greatly thickened and pulpy, and closely resembled that of a tubercular joint. I propose to thoroughly lay open this knee-joint at an early date, and deal with whatever condition may be encountered.

Case 6. *Purulent synovitis of the right knee-joint, apparently due to a leucorrhœal discharge, in a 65-year-old woman; drainage; temperature normal next day; later amputation for persistent sinuses and painful joint.*—Louisa F. was admitted to the London Hospital under the care of Mr. Jonathan Hutchinson, jun., whilst I was his substitute.

Six days before admission she was seized with severe pain in the right knee, which became hot and swollen. She had several rigors. No cause was given for this condition except that it might have been produced by kneeling whilst scrubbing floors. She had suffered from leucorrhœa for several weeks.

On admission her general condition was very bad indeed; she was in a typhoid condition. Her tongue was dry and brown, and her lips were covered with sordes. Her temperature was 102° F. The right knee was tense, tender, hot, painful, and red. The exploring needle withdrew thick pus. The knee was freely laid open and drained by four incisions—one on either side of the patella, from the inner one of which a tube passed up into the subcrureus pouch, and another across the joint below the patella. The two posterior pouches were laid open and tubes passed in through the incisions whilst the knee was flexed, which it was found relaxed the structures and enabled the tubes easily to be placed in a good position. The temperature fell to normal next day, and her general

condition rapidly improved. Her subsequent progress was not, however, satisfactory. For a fortnight her temperature oscillated from normal for a degree or two each night, but when upward massage over the calf was begun, it remained normal. This procedure evacuated a considerable quantity of pus from a deep pouch, which was probably the popliteus bursa, as in Case 8.

When Mr. Hutchinson returned to the hospital about two months after the operation, although the temperature remained practically normal, the knee was exceedingly painful, and the poor old woman showed the feeblest signs of repair. All the sinuses were open, although the discharge from the anterior ones had practically ceased. Mr. Hutchinson therefore amputated the leg in the lower third of the thigh. She made a rapid recovery.

Case 7. *Tubercular epiphysitis of the lower epiphyses of the femur; incised by a general practitioner; erysipelas; infection of the knee-joint; drainage; recurrent attacks of erysipelas; amputation.*—Victor V. B. M., a little ill-nourished child æt. 3, was admitted to the London Hospital July 28, 1902. The history was that seven weeks previously a painless swelling had appeared over the inner condyle of his right femur. This was incised by his doctor, who plugged it with gauze and fomented it.

Fourteen days before admission he became feverish and delirious, and a red mottled swelling started from the wound and spread up and down his leg; soon after his knee-joint became distended and painful. On admission a sinus was found on the inner side of his right knee which led down to carious bone. The popliteal space was greatly swollen, and contained much pus. The knee was obviously the seat of suppurative arthritis. It was drained and irrigated in the manner advocated in this paper, and in five days the temperature reached normal, and remained there except during a relapse of erysipelas. The case did not, however, do well. The sinuses remained open, and after a serious attempt to remove all the diseased bone from the carious focus in the end of the femur, which failed, but which apparently lighted up the septic mischief once more, the leg was amputated in the middle of the femur. (The specimen is shown here.)

It will be seen that the joint cavity is obliterated by fibrous tissue, and the result at the best could only have been fibrous ankylosis. In the lower femoral epiphysial line, in the situa-

tion previously occupied by the internal condyle, is a carious cavity filled with purulent tubercular material, which formed the primary focus of the whole trouble.

Case 8. *Acute pneumococcal arthritis of the right knee-joint; drainage.*—This case was under the care of Mr. Dean, who has kindly allowed me to use the case. I was substitute for him at the time.

F. D., a boy æt. 14, was admitted to the London Hospital July 30, 1902. A month previously he had suffered from influenza, from which he was convalescing, when, seven days before admission, he was seized with severe pain and swelling in the right knee, which became progressively worse. There had been no accident.

On admission the boy's condition was very bad. He was emaciated, his temperature 104°, his pulse rapid and feeble. The lungs gave no signs of existing disease. His spleen was not enlarged or tender. The whole of his right leg from the lower third of the thigh to the toes was tremendously swollen, red, and œdematous, so that it was at first a matter of doubt whether the case was one of acute periostitis of tibia with pus spreading up and down the limb, or distension of the knee-joint with pus which had extended beyond the capsule behind and involved the popliteal vein and lymphatics. The characteristic position of the knee-joint in semi-flexion, with well-marked fluctuation, decided the point. A medium-sized trochar passed under the patella failed to evacuate pus, but as it was withdrawn a gelatinous cord of pus followed it. The knee-joint was then laid open by anterior and posterior incisions. The cavity of the joint was full of clots, which so closely resembled those seen in pneumococcal empyemata that a diagnosis of pneumococcal arthritis was at once made.

The unbiassed report of Dr. William Bulloch, Bacteriologist to the London Hospital, was as follows:—Films show no organisms. Culture reveals slight growth of cocci in short chains, most probably pneumococcus.

Progress.—The temperature reached normal on the fifth day, but continued to oscillate to about 100° F. each night until the fourteenth day, when Mr. Dean, who had returned from his holiday, explored the back of the knee-joint, divided some part of the posterior ligament of the knee-joint, and evacuated a pocket of pus tracking down the back of the tibia, apparently in connection with the popliteus bursa. The

temperature then ceased to oscillate, and the boy rapidly healed up.

I think there is very little doubt that at the time of the first operation the popliteal pouch of the joint had already ruptured, and that the popliteal vessels were pressed upon. This would account for the great œdema of the leg and foot, which rapidly subsided after the first operation, and such a collection of pus, although ill-drained, was certainly found fourteen days later.

Case 9. *Acute suppurative arthritis of knee-joint occurring in the course of whooping-cough; posterior drainage.*—A little boy, W. E., æt. 5, was admitted under my care into the London Hospital, August 27, 1902. For five months he had been suffering from whooping-cough, which he still had. Two days before admission he fell, striking his knee, but producing no wound or abrasion. He had not walked since the accident.

On admission the condition of the child was very serious. His temperature was 103° F., his pulse rapid and feeble, and he was quite delirious. The knee was distended with pus, red, hot, and œdematous. The pus removed by the exploring needle was examined by Dr. Bulloch, and found to contain a pure culture of streptococci.

On August 27, 1902, the knee-joint was freely drained in the manner advocated. The temperature was normal in four days, but suppuration continued for three weeks. The tubes were removed twenty-five days after operation. His recovery was unfortunately delayed by two attacks of erysipelas, but ultimately we got him away to the country, where his sinuses rapidly healed.

He is shown here to-night with slight movement of his knee-joint, which I hope persistent massage may improve.

Some of the cases in this series are not, I admit, very happy ones, but they are, as I have said, consecutive.

The four traumatic infections, which are usually regarded as so much worse, did very well. Two had good movement; one had some movement when he left hospital; the other has fibrous ankylosis.

The five cases of blood infection were most unsatisfactory in their progress. Two had the leg amputated, but it will be seen that one was an old woman of sixty-five, who, though her temperature had been down for weeks, was unable to heal her sinuses. The other was a tubercular abscess in the end

of the femur, which became infected with erysipelas and invaded the joint.

The earlier traumatic cases had led me to hope that if the infected joint were taken in time and efficiently drained behind as well as in front, we might confidently count on obtaining a mobile joint, but subsequent cases, more especially those due to auto-infection, have made me less sanguine.

I am still confident that this method of efficient drainage will cope successfully with the immediate dangers of sepsis, septicæmia, pyæmia, and the formation of large abscesses burrowing up and down the leg, for in no case did the temperature fail to reach normal in a few days.

It is, however, clear that the subsequent progress of the cases, whether suppuration becomes chronic and sinuses persist, or whether the cartilages are destroyed and fibrous ankylosis follows, depends mainly on the cause of the suppuration. We can scarcely hope to save the leg if the disease originates from a focus in the femur or tibia.

The two most unsatisfactory cases of all arose apparently from a leucorrhœal discharge. I very much regret that no bacteriological report is forthcoming in either case.

In conclusion, I would advise that all really acute cases of suppuration in the knee-joint, say with a temperature of 103° to 104°, should be treated by anterior and posterior incisions, more especially if they arise from punctured wounds. When the suppuration arises from auto-infection and is subacute, with a temperature of not more than 102° F., I should be inclined—taught by the cases recorded above—to first of all make lateral incisions on either side of the patella and wash out the joint thoroughly and drain it. The joint must be treated as though it were an aseptic case, for in auto-infection we have usually to deal with one micro-organism only, and that often a mild one. We must therefore beware lest in casting out one micro-organism we admit several others worse than the first.

Should this partial drainage fail to bring the temperature down in a day or two, the posterior pouches should be laid open and thoroughly drained.

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Should this partial drainage fail to bring the temperature down in a day or two, the posterior pouches should be laid open and thoroughly drained.

XVI.—*Report on Sir William Gowers' Case of Local Panatrophy.* By Stanley Barnes, M.D., M.R.C.P. *Read February* 13, 1903.

AT the meeting of the Clinical Society on October 24, 1902, to which Dr. Harry Campbell showed his remarkable case of local atrophy, a short account was given of a case, in most respects similar, which I saw in private in 1885. Having ascertained that the patient was still alive, I obtained permission, through the courtesy of her medical attendant, Mr. W. H. J. Winter, of Wolverhampton, for Dr. Stanley Barnes to visit her and examine her present state. The following comprehensive report is the result.—W. R. Gowers.

As far as could be ascertained, none of her ancestors had suffered from any similar condition. Her mother and one of her sisters died of cancer, and one brother of enteric fever; she has one brother and one sister alive, the latter frequently suffering from attacks of bronchitis; all the members of the family are said to be of a "rheumatic" disposition; but, except for the patient, none of them have ever suffered from neuralgia or local wastings. Her two children are also quite healthy.

As a child the patient had an attack of "intermittent fever," which kept her in bed for about six weeks, but, apart from this, was remarkably free from disease. She lived an open-air life with plenty of exercise in the country. At the age of twenty-four she was thrown off a horse and badly bruised and shaken; but, although she was "black and blue" for a fortnight, she was not laid up in bed. A second accident occurred a year later; she fell on to the back of her head whilst skating, and is said to have been unconscious for a quarter of an hour, but was able to walk home. She is quite certain that as a child there was no atrophy such as is now present.

At the age of thirty-two, or thereabouts, she began to have intermittent neuralgia, which, beginning in the left

supra-orbital region, rapidly spread until the supra-orbital and infra-orbital regions on both sides of the face were involved. The neuralgia was always worse just before the onset of menstruation, ceasing, as a rule, when the discharge came on. The neuralgia increased in intensity and in constancy for several years, despite the removal of all the upper teeth; it was never sufficiently severe to prevent her from doing her household work for any long period, and with the onset of the climacteric, which is not yet fully established, at the age of fifty-two, the neuralgia has greatly lessened, but has not ceased.

When thirty-four years of age she first noticed that she could not raise the left arm so well as usual, and this weakness became more and more pronounced for the next two months; a friend then happened to see the shoulder and called the patient's attention to it, remarking that she must have dislocated it. A medical man was called in, and found that there was extensive atrophy of the deltoid, but that there was nothing wrong with the shoulder-joint. Shortly after this the patient was seen by Sir William Gowers in consultation, and other patches of atrophy were found. As far as the patient can tell, with the exception of those on the head and neck, all of the patches now present were found then; nor have any of them undergone any material alteration. A little improvement took place in the left shoulder muscles at first under a course of massage and galvanism, but no further change has occurred there in the last sixteen years.

Ten years ago, when she was forty-two years old, she noticed that her nose was not quite straight, that it was turned a little to the left; she feels sure it used to be all right before that. No further change occurred until at the age of fifty the right side of her mouth began to act badly; this weakness became progressively more pronounced for twelve months, by which time the right side of the face was almost if not completely paralysed, so that she had to use her hand to close the right eye. This facial palsy has since then slowly improved, and her husband and the patient agree that it is now hardly noticeable some days, whereas if she is agitated—as by a clinical examination—it becomes very obvious. Since the facial paralysis came on patches of atrophy of the skin have been noticed on the face; with one exception these patches have not extended for some months now. For the last few days there has been some quivering in the right levator menti region, where a dimple has developed quite

recently; this seems to be the latest developed of any of the patches of atrophy.

Pari passu with the facial palsy came on a difficulty with memory; she would often hesitate about finishing a sentence, having forgotten what she had already begun to say; this is quite well now.

Present condition.—The patient is a well-educated women of fifty-two years of age. The general health is good, the viscera show no signs of disease, and except in particular places the muscles and subcutaneous tissues are well developed. There is no shortening or general atrophy of any limb or of any large group of muscles; nothing to suggest an old infantile cerebral or spinal palsy can be found.

Smell, taste, and sight are good; she hears a watch as well with the right ear as with the left, and never suffers from tinnitus. The pupils are equal, of normal size, and react well to light and on accommodation; they dilate normally on shading the eyes. There is no defect of sensation on any part of the face; the bones of the face and skull are well and symmetrically developed. There is an almost complete right facial palsy of the peripheral type, with very slight contracture. Every muscle supplied by the right facial nerve is about equally affected, only slightest actions being possible. She cannot close the right eyelids, but can keep them closed when they are brought together for her. She "used to be always whistling," but now cannot whistle at all.

Electrically all of the right facial muscles respond to faradism, but less well than on the opposite side; they require a stronger current and give a feebler contraction. Galvanism was not tried. There is only one small patch of atrophy of the skin on the right cheek, and one very small one on the right chin. On the left side of the face patches of skin atrophy are also present on the forehead and cheek. Apart from these the face in repose is almost symmetrical, but on smiling the right side moves very slightly and the left very markedly.

The condition of the face seen here in no wise resembles what I have seen elsewhere in cases of facial hemiatrophy; it rather suggests an ordinary Bell's palsy which is improving.

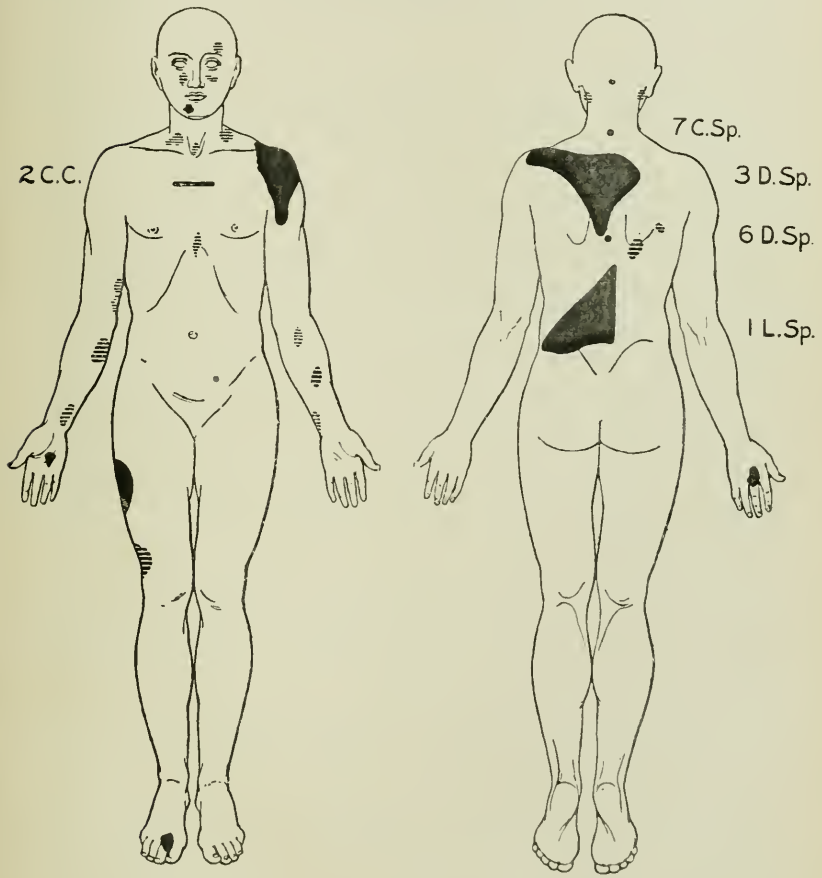
A small atrophic patch of skin is present over each stylomastoid region, but the skin was freely movable, and it did not appear that the facial nerve was here involved in any cicatricial process. The patch is no bigger on the right

than on the left, and yet the left facial muscles are normal and the right are almost completely paralysed.

There is no atrophy of the tongue or buccal mucous membrane.

Motor system.—Atrophic muscles only are weak, and weakness corresponds directly to atrophy throughout.

Fig. 4.



The following muscles are wasted in patches, as shown in the diagram:

On the left.—Deltoid, supra-spinatus, infra-spinatus, rhomboid, erector spinæ.

On the right.—Second dorsal interosseous hand muscle, lumbricales, erector spinæ as in the diagram.

It is very clear that where the muscles are wasted the disease has invaded muscular tissue corresponding to areas of atrophic skin as though by a direct punching-out process, so that only a part of a muscle may be affected. This is very clear in the left deltoid, where the outer and posterior parts of the muscles are good, whilst the anterior part is severely atrophic. The same is also very marked in the left erector spinæ muscle.

All muscles present respond normally to mild faradic currents, and as well as the unaffected muscles, though of course less powerfully. The reflexes are normal.

Skin, etc.—There is *absolutely no anæsthesia* on the patches or elsewhere.

Nowhere is there any leucoderma or any pigmentation beyond the normal. In the areas affected (*see* diagram) the skin is smooth, hairless, shiny, and not adherent to the deeper tissues. It is remarkably thin, and can easily be pinched up and rolled between the finger and thumb. In a few places, especially on the back, it is wrinkled and puckered, but this is evidently due to redundancy and not to fibrous adhesions.

In several of the patches, particularly the smaller ones on the forearms, neck, and face, there is no atrophy of subcutaneous tissue. But in those on the back, the left shoulder, the right thigh, hand and foot, the subcutaneous fat has almost disappeared, the superficial veins, however, remaining well preserved.

Muscle atrophy accompanies the skin atrophy in all the larger patches. In only one place is *bone atrophy* definite, viz. the second dorsal spine. The middle phalanx of the right second toe is bared and feels small; it is doubtfully atrophic. The scapulæ are normal in size.

Throughout the patches have a sharply punched-out appearance, the line of junction between healthy and diseased skin being a very abrupt one.

The subcutaneous tissue immediately surrounding a patch, when rolled under the finger, gave the impression of being unusually lobular, but this might easily be due to the thinness of the skin readily allowing of the finer details of the adjacent tissue being easier felt here than elsewhere.

XVII.—*Two Cases of Prolapse of the Bowel, and a Case of Prolapse of the Uterus, treated by the injection of Paraffin under the Mucous Membrane.* By Stephen Paget, F.R.C.S. *Read March* 13, 1903.

In 1900 Gersuny published his first set of cases treated by injection of paraffin, and he then suggested that the treatment might be useful in cases of prolapse. That same year he published the first case of this kind; it was that of a woman suffering from vesico-vaginal fistula, with incontinence of urine; she had already undergone seven operations in vain. Gersuny cured her by injecting paraffin under the prolapsed mucous membrane of the bladder, so that a sort of valve was formed which prevented the leakage of urine. I will not take up the time of the Society by reviewing the whole history of the paraffin treatment of cases of prolapse. I will only say that the number of recorded cases is so large that there is no element of originality in my own results. I have used the method in three cases only—two of prolapse of the bowel and one of prolapse of the uterus. The first case has already been published, so I will say very little about it. The patient was a thin, feeble man sixty-five years old. Six years ago he had undergone the operation of excision of the rectum for cancer. Ever since that time he had suffered prolapse, and had worn a plug. Sometimes the prolapse would come down in spite of the plug, and he would be soiled with the discharge from the bowel; and always, if the bowels were relaxed, or if he took hot food or hot drink and then was unable to go at once to the closet, he would be soiled; and the constant wearing of the plug was, of course, a great discomfort to him.

Examination showed the opening into the bowel lying at the apex of a deep, cup-shaped, cicatricial fossa; the ring round the lower end of the bowel was lax, and easily admitted three fingers, and there was no trace of a sphincter. While he was in bed there was no prolapse; when he walked about, or bore down gently, about two inches of mucous membrane were everted.

On January 15 I injected paraffin at several points into

and around the prolapse, and raised two hard round nodules under the mucous membrane, so that the bowel would admit only one finger. One of these nodules or hummocks of paraffin lay just inside the anal ring, and had the appearance of blocking it right across, but was easily pushed aside with the finger; the other nodule lay a little higher up. Thus a sort of valve was formed. The result was very good; he left off his plug, and the bowel did not come down, and he kept perfectly clean. I examined him on February 25, six weeks after the injection, and he told me that the bowel never came down, and that he had no discharge or trouble of any kind from it. The two nodules of paraffin were not altered in any way, except that they were perhaps a very little flatter than when he had left the hospital. I examined him again this afternoon and found him still perfectly comfortable. Probably he will require, sooner or later, a little more paraffin; but, for the present, there has been no sign of prolapse, and no leakage from the bowel.

The second case was that of a patient of Dr. Lycett Burd, of Shrewsbury. She was sixty-one years old, and had suffered from prolapse of the bowel for twenty-two years. She had undergone five operations for it, and had then been advised to have nothing more done, but to lead a semi-invalid life. Prolapse occurred on the least exertion, such as lifting a light weight or sneezing; there was sometimes a slight bleeding or an escape of flatus; and, if the bowels were relaxed, she could not control them. There was also some incontinence of urine. The external sphincter ani seemed to act well; but, when she bore down, about three inches of mucous membrane were immediately shot out. The opening into the bowel was lax, and easily admitted two fingers.

On February 15 I injected paraffin under the mucous membrane of the prolapse, close to the junction of skin and mucous membrane, or just above this level, making a hard, raised collar of paraffin round half or three fourths of the circumference of the bowel.

On March 1 I repeated the injection, because her condition, though it was greatly improved, was not perfect; there had been more than once a slight prolapse, but not half what it used to be. On this second occasion there was no prolapse under the anæsthetic; and I had to hook down the thickened bowel, and, as it were, to deliver it through the anal ring. I used a considerable quantity of paraffin for this second injection, nearly an ounce and a half, so that the lower two

and a half inches of the bowel felt all thick and hummocky. Then I narrowed the anal ring by the incision and suture described by Mr. Mayo Robson in the *Practitioner* (February, 1903). I am sorry to say that she had severe pain after the injection of the paraffin, and still has attacks of pain at times—for instance, after an enema. The bowels were kept quiet till March 6, and then there was a good deal of difficulty in getting them to act; but with a dose of castor oil they acted rather too freely, again and again, for six or seven hours. There was no prolapse. I hear to-day that she remains free from the least sign of prolapse. Two of the superficial stitches of Mayo Robson's incision, nearest the rectum, have cut out; otherwise the little wound has healed well. Unhappily she still has some pain in the rectum, and is still obliged to use the catheter. I hope that these troubles will cease now that she is no longer kept in bed.

The third case is that of a woman aged 63, who had suffered for twenty-eight years from prolapse of the uterus. She wore a pessary at one time, but that was twenty years ago. She underwent an operation at the Liverpool Southern Hospital fourteen years ago, which seems to have been Alexander's operation. The prolapse occurs even when she is lying in bed, if she merely coughs or sneezes, or turns over in bed; and for the last six months it has been down all day and every day, just supported in a napkin.

Examination showed complete prolapse of both vaginal walls, together with the cervix uteri, which was greatly enlarged, about half the size of one's fist, and was covered with coarse, dry mucous membrane looking like the skin of the scrotum. The vaginal orifice was so much dilated that it would admit the whole fist, and no form of pessary could be retained. The perineum had been ruptured up to the edge of the sphincter ani.

On February 25 I injected paraffin in various directions under the mucous membrane of the vagina and the cervix, wherever it seemed most likely to support the prolapse and to narrow the vaginal orifice. Altogether nearly an ounce and three quarters of paraffin were used. The injections under the mucous membrane of the cervix were mostly made into its posterior portion; but a small quantity of paraffin was also injected into its anterior portion, care being taken to keep away from the immediate neighbourhood of the bladder and the urethra. The injections into the posterior vaginal wall were guided by a finger put up the rectum.

Five days later, March 2, the notes say that there has been no sign of the least prolapse since the operation, and the patient says that she feels more comfortable than she has felt for years. She moves about freely in bed, coughs, sneezes, etc., and nothing comes down. Examination shows two chief masses of paraffin in the vaginal walls; that in the posterior wall feels like a rounded hard buttress, as thick as one's thumb, all the way up the vagina. There is marked thickening and hardening of the cervix; the antero-posterior diameter of the vaginal cavity is reduced by a third, more or less; and the whole cavity feels set and rigid.

A day or two later she was put to various tests, including the crucial test of walking to the closet and there getting rid of a very constipated motion; and there was no prolapse.

On March 7 she was examined by my colleague at the West London Hospital, Dr. Drummond Robinson, to whose kindness I owe the case. Dr. Robinson makes this note on her condition: "There is no prolapse of the vaginal walls, even when the patient makes the strongest straining efforts. *Per vaginam*: The cervix uteri is high up. The posterior vaginal wall feels indurated; the recto-vaginal septum is about half an inch thick; the anterior vaginal wall is indurated." And he adds, "This was about as bad a case of prolapse of the vagina and uterus as you could have selected." The patient left the hospital yesterday, perfectly well and comfortable.

I venture to bring these cases before the Society thus early because I am sure, from my experience of the use of paraffin in forty cases of depressed nose, that the improvement in these prolapse cases will be well maintained. If it ever should be necessary to add a little more paraffin, this could easily be done.

Note.—*June*, 1903.—The two cases of prolapse of the rectum remain well, and free of all trouble. The case of prolapse of the uterus, though it was greatly improved, was not perfectly cured, and about the end of April I made a second injection, and also repaired the perineum. She remains well, and free of all trouble.

XVIII.—*Case of Total Paraplegia due to Caries of the Mid-dorsal Vertebræ, for which the operation of costo-transversectomy was performed, with complete recovery.* By W. H. B. Brook, M.D. *Read March* 13, 1903.

ARTHUR A., æt. 17, clerk, was admitted into the Lincoln County Hospital on September 22, 1902, complaining of loss of power in the legs of four weeks' duration.

History.—In November, 1901, he noticed a lump on his spine in the mid-dorsal region, which was very painful, the pain, however, disappearing after two weeks' rest in bed. After this he resumed work until May, 1902, when he had to give up entirely. He wore a plaster jacket from March to May, since which date he has been confined to his bed. During the last four weeks he has lost the use of his legs, and has had difficulty in retaining his urine.

Family history.—No history of phthisis or any form of tuberculosis.

Condition on admission.—He is well nourished, but pale and anæmic. There is an angular curvature of the spine in the mid-dorsal region, the sixth and seventh spinous processes being the most prominent. Percussion over the projection causes no pain, and there are no signs of any abscess pointing. There is a "girdle sensation" at the level of the epigastrium. Sensation below this level is much impaired, but not totally absent. He can move the legs, but not well. The legs are spastic, but there is no atrophy of the muscles. He has incontinence of urine, and to some extent of the motions if the bowels are relaxed. The knee-jerks are exaggerated; the Babinski phenomenon was not present. Nothing abnormal was discovered about the heart or lungs.

Urine.—1030, acid; no albumen; no sugar; phosphates.

A seven-pound extension was applied to each leg.

He was ordered a mixture containing phosphate of iron, with cod-liver oil and malt extract.

November 7, 1902.—During the six weeks that have elapsed since his admission his condition has become steadily

worse. He has now complete paraplegia; sensation is totally lost in the right leg, almost so in the left. The knee-jerks are much exaggerated, and violent ankle-clonus is easily obtained. Babinski's phenomenon is present in both feet. Urine and faeces are passed unconsciously. There is a girdle sensation at the level of the seventh rib, but no tenderness on percussion over the prominent spinous processes. In spite of the most careful nursing there are sores on the back of each heel. Temp. 98·4° to 98·8°. A skiagraph was taken which showed blurring and "halation" in the region of the seventh and eighth dorsal vertebræ, which confirmed the diagnosis that tubercular matter was present, causing pressure upon the cord, and it was decided to attempt to relieve this by operation.

November 8.—Under chloroform, a transverse incision three inches long was made, commencing to the right of the sixth dorsal spinous process, slanting slightly downwards to the right. The muscles were cut through until the right transverse process of the eighth dorsal vertebra was exposed. This was cut through at its base and removed. The periosteum of the eighth rib was incised from the head to just external to the tuberosity, and was then carefully stripped from the rib, which was cut through and its head and neck removed. Whilst this was being done thick curdy pus welled up, and it was found that the head of the rib was carious, and detached from the body of the eighth vertebra, which was practically destroyed. A sharp spoon, made specially for the purpose with a curved shank, was introduced, and all the soft bone scraped away as far as possible, care being taken not to wound the dura mater or the pleura. A drainage-tube was inserted, and iodoform emulsion containing 1 in 40 carbolic acid was injected, and the cavity lightly packed round the tube with iodoform ribbon gauze. The patient was carefully placed in bed, the mattress of which was in several sections to facilitate dressing, and a seven-pound extension applied to each leg.

November 9.—The wound was syringed out with carbolic lotion 1 in 40, and iodoform emulsion injected; there had been a profuse discharge of curdy pus. There is a slight return of sensation in the right foot. The right leg keeps jumping up in spite of the extension, to the great discomfort of the patient.

November 10.—There is a "pins and needles" sensation in the right foot.

PLATE III.

Illustrating Dr. Brook's Case of Total Paraplegia due to Caries of the Mid-dorsal Vertebræ.

A skiagram showing the situation of a mass of tubercular material in connection with spinal caries.

Bale and Danielsson, Ltd.

November 11.—Dressed, and iodoform emulsion injected; still considerable discharge. Is able to move the great toe of each foot.

November 29.—There has been a steady improvement from day to day. Sensation is perfect, and the patient can move his legs freely. The spasmodic involuntary contraction of the muscles is less troublesome, but there is still incontinence of urine and fæces.

December 27.—The patient continues to improve; he now knows when he is about to pass urine and fæces, and can retain them till he is attended to. He can lift each leg freely from the bed, and his sensation is perfect. The sores on the heels, which were very troublesome before the operation, have rapidly healed. The girdle pain has entirely disappeared. The wound is still dressed every other day, and iodoform emulsion injected, the tube being retained. There is very little alteration in the curvature; the legs do not now jump much, but ankle-clonus can still be readily obtained. His general health and nutrition are all that could be desired; he eats and sleeps well. Since the operation the temperature has never reached 99°.

January 8, 1903.—The tube was discontinued.

January 28.—The wound has gradually granulated up and is now quite healed; sensation is unimpaired; he can move both legs with ease. He has no paræsthesia, and no girdle sensation. There is perfect control over the bladder and rectum. He has put on flesh and is in excellent health.

February 4.—Fitted with a steel spinal support, consisting of a pelvic band and two dorsal rods one inch apart so as not to press upon the spinous processes, with shoulder straps, and plates so arranged as to support the spine where it is defective, whilst applying at the same time a certain amount of extension.

February 15.—Allowed to sit up for an hour daily, wearing the apparatus; the Babinski phenomenon has disappeared. Pulse 88; resp. 20; temp. 98·4°.

February 19.—Walked for the first time since his admission.

March 13.—Is able now to walk quite well.

I have ventured to bring this case before your notice as an example of a comparatively recent method of treating a not infrequent complication of a very common disease—pressure paraplegia caused by spinal caries; and I am the

more encouraged to do so as I first learnt this method by seeing a case shown here last October by Mr. Jackson Clarke, to whom, for his kind advice and suggestions, I am very greatly indebted.

There are certain points which are perhaps worthy of consideration. On first seeing the patient it was difficult to decide whether the paraplegia was due to compression of the cord by the broken-down bodies of the diseased vertebræ, or whether it was due to a pachymeningitis, as there was no sign whatever of any collection of pus.

The diagnosis, however, was assisted by two points:—(1) There was no tenderness whatever on percussion over the prominent spinous processes, which one would have expected had there been any pachymeningitis. (2) The skiagraph showed a blurring or "halation" extending for a radius of two inches at the site of the disease, an appearance extremely suggestive of the presence of caseous deposit. These two points inclined me to think that I had to deal with a pressure paraplegia due to the presence of caseous matter outside the dura mater.

As to the treatment, it was suggested that a laminectomy might give relief; this, however, I was unwilling to do, as it would have deprived the patient of the support of the only part of the vertebral column which at the level of the disease was still sound—namely, the laminæ and spinous processes. I therefore tried what could be done by rest and extension, but without any benefit, the patient's condition steadily deteriorating.

It was at this time that I saw shown here Mr. Jackson Clarke's successful case, which decided me to adopt the same treatment. There was no particular difficulty about the operation, which was rendered all the easier by the head of the rib being already detached from its vertebra. Care had, however, to be taken not to injure the spinal cord on the one hand or the pleura on the other.

It is interesting to note that sensation began to return within twenty-four hours, and voluntary movement within forty-eight hours, but that it was not until six weeks had elapsed that control over the bladder and rectum was regained, long before which time the patient could move his feet and legs perfectly. The disappearance of the Babinski phenomenon is also of interest.

I have ventured to bring this case before you in the hope that it may lead to the more general adoption of this method

of treatment, presenting as it does manifest advantages over the more usual operation of laminectomy. I have to acknowledge my indebtedness to Dr. Kinmont and Mr. J. H. Crowe, the house surgeons to the Lincoln County Hospital, for the great care with which they watched the case and carried out my instructions.

XIX.—*On Skin-sliding as a means for Preventing and for Removing Cicatricial Deformity.* By T. S. Ellis, of Gloucester. (Communicated by the President, Mr. Howard Marsh.) *Read March* 13, 1903.

In the process which I have devised for preventing and for removing cicatricial deformity, my object is to utilise that property of cicatrix or scar tissue by the operation of which the deformity is caused. The disposition to contract, very manifest in a cicatrising wound, less so in a cicatrix a long time formed, would be equally effective in every direction, and the contraction would continue to draw the surrounding skin towards it if only the margin remained movable. This, however, becomes firmly adherent to the subjacent tissues, thus protecting the skin outside it from any further traction.

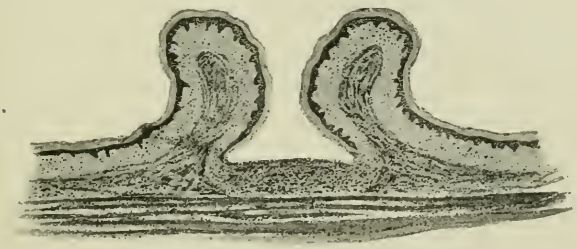
The course of events may be represented in diagram thus :—Let vertical arrows, pointed downwards, represent the force which draws the cicatrix towards its base, and horizontal ones that which draws it towards the opposite side of the wound, but is insufficient to entirely overcome the adhesion at the margin of the cicatrix. Then, in accordance with the law of the parallelogram of forces, the resultant of these two components, which may be expressed by other arrows placed obliquely, produces a depressed cicatrix of an area rather less than that of the wound, and fixed to the subjacent parts.

My principal purpose in this paper is to point out that, in a cicatrising wound, if adhesion at the margin be prevented and the adjoining skin be kept movable, we may get the full benefit of the force acting horizontally, which will draw the skin over the area occupied by the wound. This object may be accomplished by persistent sliding of the skin towards the wound, and, if this skin be made freely movable, it can be used as a handle for uplifting even a firmly adherent cicatrix (Fig. 5). The cicatrised margin of the wound, thus uplifted or loosened, being free to contract in every direction, effaces itself by contraction, and, in doing so, compels the adjoining skin to slide into and occupy its place; while we can rely on growth in the structure of the extended skin to compensate

for the stretching it has undergone. This is a fund upon which we can, literally, *draw* to an indefinite extent, for, very unlike a banking account, it recuperates itself. Thus the area of the cicatrising wound is, to the extent of the uplifted or loosened margin, diminished, and, the process being continued, the whole is ultimately replaced by skin. The surgeon's part in the proceeding—to draw or to push the skin towards the cicatrising wound or cicatrix—is an act of sliding; and Nature's part—to draw the skin into the area of the cicatrising wound—is also a sliding action. Hence the term which I have applied to the process—skin-sliding.

The necessary manipulation does not cause any severe pain. Naturally the patient shrinks, at first, from touching of the skin near a wound; afterwards it is hardly noticed. On the other hand, the wound is more comfortable when the cicatrix

Fig. 5.



is not adherent. The aggregate amount of pain is, I am sure, diminished by the proceeding.

I may add that when the surface is closely adherent to subjacent parts, as on the face, the palmar aspect of the fingers, or over the front of the tibia, and it is difficult to hold the skin for the purpose of making it slide, the process is much facilitated by using a piece of granulated india-rubber, such as that used in Hancock's washing-pads. This holds the skin to be moved, and, at the same time, is held by the fingers pressing upon it.

To myself the following case would be one of much interest, if only as an instance of successfully applied theory. I certainly had no experience of any large wound that had been healed with a covering of skin, but I had seen enough to give me full confidence that the principle which I have just formulated would hold good.

Ada G., æt. 10, was admitted to the Gloucester Infirmary on January 15, 1902, with severe burns. The integuments of the neck had been destroyed; both ears were almost entirely effaced. I offered Mr. Washbourn, whose case it was, to take charge of it as a case to test the value of skin-sliding, and I promised that no deformity of the neck should remain. This promise has been fulfilled; but I ventured also to predict that no induration would remain in the line of the scar—nothing in the nature of keloid,—in which also I hope to show that I have been justified, although, at first, it was only so as regards the lower margin of the wound. This margin, during the process of healing, was kept free from attachment to the parts beneath by lifting the skin with the finger and thumb each time the wound was dressed, and it healed in a continuous, sharply defined line, the skin being perfectly smooth and normal. The provision of new skin has been mainly due to stretching and growth near the margin; this is shown by the small tattoo mark near the margin, and the relative position of it in the second photograph. The upper margin of the wound, on the cheeks and above the chin, was, however, left to itself, for fear—a mistaken fear, I am sure—that the skin of the face would be drawn downwards. On this, the upper line, keloid formed, as it did on both hands and on both shoulders, also badly burnt. The child left the hospital six months after the burn, when all the wounds had healed. The condition of the face was then very much the same as seen in the second photograph, taken three months later, except that the keloid increased in thickness along the upper margin of the scar on the face, while the lower margin of skin continued to advance upwards so that it was overhung by the keloid above it. Keloid formed everywhere, except in the one wound, but in this one it was only along the margin which was not treated by skin-sliding. The striking contrast between the upper and the lower margins of the wound cannot be explained by the irregular line left by the upward-directed flame, as against the more even line of destruction by heat on the lower margin. No one could suppose that keloid forming under ordinary conditions would have the sharply defined edge seen on the lower margin. The child returned to the infirmary on October 8. The keloid on both sides of the face has since then been excised, and both margins of the resulting wounds treated on the same principle; the margins were not allowed to become adherent. The result is a much narrower line.

The chin has been left for the present. For this, and it may be for the cheeks, further operation will be necessary. When the skin has been made to be more extensible there will be no difficulty in bringing the edges together.

Here I venture to digress with a remark on cicatrices generally. My confidence that, ultimately, no conspicuous scar will remain in this case is based on considerations entirely opposed to the theory of Langer's lines (Langer'sche Linien) as set forth in Kocher's *Operative Surgery.* My observations show me that cicatrices of wounds made in the long axis of the body are not as completely effaced as when they accord with the transverse foldings of the skin. I think that too much is made of the coapting effect of extension in a longitudinal wound, while the widening effect of relaxation, or relative relaxation, in the position of rest is disregarded. It is true that in a transverse wound there is a widening in extension, but there is a complete coaptation when the part is relaxed, and it is because the skin along the ramus of the jaw has, in relaxation, the line of folding nearly in accord with the line of the cicatrix that I predict a very narrow line of scar when the case is complete.

No photograph of the hands was taken until eight weeks after the burn. The skin on the dorsal surface of both wrists and of both hands and fingers had been destroyed, including, as far as the roots of the fingers, most of the extensor tendons. On the right wrist the skin of the palmar surface was included, forming a complete ring. The final phalanx of the left thumb dropped off with the dressings. Such wounds could at first only be left to cicatrise in the ordinary way, but when the child had so far recovered that we felt free to manipulate the skin of the arm as well as that of the neck, the margins of the cicatrising wound were kept as movable as we could make them. Here, too, thickened and contracted cicatrix tissue has been excised, the skin undermined, brought as far as possible downward, and treated by skin-sliding. The result is, so far, satisfactory, but the proceeding has to be continued.

This plan of operation would, I feel sure, be applicable to any case where, although the area to be covered is large, skin might be drawn from both sides, and the condition shown in Fig. 5 might be realised before operating. It is better to have repeated operations than to attempt to cover a very wide area at once, but so far as I can judge, if there be movable skin in the neighbourhood, there is hardly any

limit to the area which may be covered by the process of skin-sliding.

Where a cicatrix is fully formed and is adherent to adjacent parts, it may be lifted from its attachment by persistent movement of the skin in the manner indicated in Fig. 5, and when the skin has become freely movable it may be inverted and pushed beneath the uplifted margin of the cicatrix, thus still further uplifting it. When a narrow cicatrix is so far uplifted as to admit of subcutaneous section, this proceeding is clearly indicated; it is not always necessary. Sufficient disposition to contract may remain in the cicatrix, so that, being detached and free to contract, it will then efface itself. A cicatrix-clamp to press together the skin on either side behind the cicatrix is very useful. I have recently shown a case, that of Miss M. H., where an adherent cicatrix followed gangrene of flap with ulceration of cartilage after a Stephen Smith amputation at the knee. This cicatrix, a year after its formation, was completely lifted from the bone by persistent application of the principle indicated in Fig. 5. The end of the bone is now covered by freely moving skin with only a narrow line of cicatrix, hardly perceptible. On this skin at the end of the bone the weight of the body is borne, as I think it ought to be in every stump after amputation. In the so-called "bucket" form of artificial leg, which takes the bearing on the sides, the weight of the body drags the skin away from the cicatrix. This seems to be wrong; in practice it is certainly not comfortable.

These are the grounds on which I base my plea for some reconsideration of the principles governing the treatment of cicatrices and of cicatrising wounds.

XX.—*Two cases illustrating the Employment of Invagination and Stitching over Gangrenous Patches of Intestine in Strangulated Hernia.* By G. H. Makins. *Read March* 27, 1903.

THE following two cases are related as of interest as bearing on a condition of very great importance, while the method of treatment successfully adopted does not appear to have obtained the consideration it perhaps deserves.

Case 1.—R. B., æt. 87, a shoemaker. The patient, a feeble old man, had been the subject of an irreducible right inguinal hernia for sixteen years. Twenty hours previous to his admission, in June, 1898, he had been attacked with severe pain in the hernia, he had vomited several times, and the bowels had been confined. The tongue was somewhat dry, but the general condition fair considering the age of the patient. The scrotum was considerably swollen, red, and very tender. The hernia was obviously strangulated.

A herniotomy was at once performed. On opening the sac a quantity of stinking bloody fluid and a single apple pip escaped. About six inches of small intestine were found in the sac, red and inflamed. On manipulation, gas and fæcal contents escaped from the gut, and on examination a small patch of gangrene was found on the convex margin of the gut, and a small perforation.

The condition of the patient was not such as to allow of a serious operation; the small gangrenous patch was therefore treated by invagination with four Lembert's stitches, the bowel was washed, the neck of the sac divided, and the bowel replaced just within the internal ring. No attempt was made to close the wound, but this, as well as the large sac cavity, was plugged with gauze.

The patient made an uneventful recovery, the wound granulating up slowly, and on the forty-sixth day he left the hospital cured.

Case 2.—J. M., æt. 9 weeks. An ill-nourished illegitimate child, the subject of congenital syphilis, and being brought up by hand, was brought to the hospital in June, 1902.

On the day previous to the child's admission the child was fretful, and the nurse noticed the scrotum to be red and swollen. The child's bowels acted, and there was no vomiting.

On admission the scrotum was found to be red, swollen, and tender, with considerable œdema. Manipulation was very painful, but revealed slight fulness at the right internal abdominal ring, and this spot was manifestly tender.

The child was anæsthetised and an incision made into the scrotum. A small hernial sac containing foul-smelling bloody fluid was opened, and at the bottom of this was found a partial enterocele of the small intestine. On releasing the gut the strangulated area, about three quarters of an inch in diameter on the convex border of the bowel, was found to be gangrenous. The bowel was greenish black, flaccid, and there was entire loss of elasticity and tone. This patch was invaginated by the insertion of four Lembert's stitches passed from the healthy margins across it. This procedure caused an angular bend of the gut, but pressure showed that a free lumen persisted for the passage of the contents of the intestine. The gut was washed and returned just within the internal abdominal ring, which was left freely open. The sac was then removed and the wound plugged with gauze.

The bowels acted freely soon after the operation, and the infant made an excellent recovery without any untoward symptoms. He left the hospital at the end of fourteen days.

I have seen the child from time to time since, on the last occasion in February, 1903. He has been under treatment for the congenital syphilis, has developed well, and there has been no trouble with the bowels.

I have twice followed this method in herniæ in which there was reason to question the viability of the gut at the actual seat of constriction, with a good immediate result. I do not, however, wish to place much weight on these, because I am unable to say that the condition was more than a highly doubtful one. Of a fifth case, in which a patch of gangrene due to partial enterocele was invaginated, I unfortunately kept no record beyond the fact that the immediate result was successful.

The two cases above related, however, serve to raise the subject of the value of the method. I have particularly chosen the first because it was the experience gained from it which suggested the method to me, and encouraged me to give it a further trial. I must, however, at once admit that

the extremely local nature of the gangrene rather places the case in the category of perforation of the small intestine than in that of gangrene of a strangulated hernia.

The second case, however, was an undoubted instance of gangrene of a partial enterocele. Failing the procedure adopted, no choice would have remained but to establish an artificial anus, or resect the bowel. The dangers of the former alternative are familiar, and the latter, in view of the feeble condition of the child and his tender age, would in my opinion have been certainly fatal.

I am unaware whence this method originated; one of my own colleagues, Mr. Robinson, I know occasionally makes use of it, but it does not, as a rule, gain mention in the classical English text-books.

In Cheyne and Burghard's *Manual of Surgical Treatment*, 1902, p. 433, vol. vi, the following passage occurs:—"Where there are only one or two suspicious spots on the bowel, and it is decided to return the latter, it is well to invert the suspicious area by Lembert's sutures, so that should they become gangrenous no perforation will take place into the abdominal cavity."

In the *Centralblatt für Chirurgie*, No. 10, 1903, p. 298, reference is made to two cases recorded by G. J. Breitmann: —"Like Arapow and Kadjan, Breitmann has twice invaginated gangrenous areas in the wall of the colon into the lumen of the gut in treating strangulated herniæ. The areas affected measured six by two centimetres, and perforation had occurred in the centre of them. Both cases recovered, in the second after the development of slight peritoneal symptoms which persisted for two days."

In the article on hernia by Prof. E. Graser in the *Handbuch der praktischen Chirurgie* (von Bergmann, von Bruns, and von Mikulicz), vol. ii, p. 984, 1902, the following judgment is pronounced upon the method:—"The method of invagination and stitching over gangrenous patches or doubtful areas, especially those at the seat of constriction, which has been successfully performed and highly recommended, has proved itself so dangerous that it is better sternly avoided. In three of five cases in the Heidelberg Klinik in which it was adopted fatal perforation occurred in the proximal portion of the gut above the seat of constriction. Again, where more extensive invagination is required stenosis is to be feared. In such cases resection is to be preferred."

With this latter opinion I venture to join issue, and I

think that the recommendation of Cheyne and Burghard may well be extended from the grade of suspicious areas to that of pronounced gangrene in suitable cases. It is of course clear that when the condition of the bowel is one of diffuse infective inflammation extending above the seat of constriction, or when the duration of the strangulation or the appearance of the gut suggests the presence of ulceration above the point of obstruction, local invagination would be a hopeless measure. On the other hand, when we are fairly confident that the gangrene is purely mechanical in origin, local, and not accompanied by diffuse infective processes in the upper segment of the bowel, I believe the method of invagination to be both useful and safe.

As to consecutive stenosis, I do not see that its chances are greater after sinking a gangrenous ring than after an enterectomy with union by means of a Murphy's button; while we are aware that strictures sometimes follow a simple circular enterectomy, or even develop in portions of strangulated bowel apparently recoverable, which have been returned with good immediate result.

The two cases above related, moreover, appear to me to demonstrate that inversion and stitching over may be applicable where the method of primary resection, now so steadily gaining in favour, may be impracticable consonant with the recovery of the patient.

XXI.—*A case of Bilharzia Disease.* By Andrew Duncan, M.D. *Read March* 27, 1903.

I VENTURE to bring the following case before you, as we are likely in the immediate future to see cases of this disease in the persons of officers and men returning from South Africa, so that the questions of diagnosis and treatment are of some importance. The case I am about to relate also presents one or two features of interest.

H. M., æt. 30, late of the 2nd Battalion East Kent Regiment, went to South Africa in January, 1900. He was fighting there in the Orange River and Cape Colonies, and was in Lord Roberts's march to Pretoria, after which for some months he was engaged in the Orange River Colony and Transvaal, and, finally, was at Nelspruit in June, 1902. In May, 1902, he began to experience itching at the end of the penis and a frequent desire to urinate, with great straining, but had at this time no hæmaturia. The above symptoms were at their worst in the middle of the day. During this time he had apparently a sympathetic glandular affection in the left groin, as the "lump" went away after rest; great pain in the back was also experienced.

He returned to England in June, 1902, and then on arrival he first noticed a small clot of blood in his urine. Previous to the appearance of this clot he had had severe pain and burning in the urethra. Soon after the passage of the clot more blood passed mixed with the urine. In July, there being a decided increase in the hæmaturia, he went into a country hospital, and was there sounded for stone and examined by the Röntgen rays. At this period growths appeared around the anus, for which mercury was prescribed on the supposition that they were specific. In January of the present year he presented himself at the Polyclinic, where he was seen by my colleague, Mr. Cantlie, who at once recognised the case as one due to bilharzia, and sent him to the Branch seamen's hospital, where he was admitted under myself.

On admission his condition was as follows:—His temperature was subnormal; he was anæmic. The urine was

acid, sp. gr. 1022, and contained blood, mucus, and a slight amount of albumen, with numerous ova of *Bilharzia*. The blood was passed at the end of the act of micturition. In the perinæum on admission there was a slight swelling over the raphe, which disappeared after being fomented. There were three growths around the anus—one conical, of about half an inch in diameter at its base; the other two flattened, with broad bases half an inch in diameter and 1 and 1½ inches in length respectively. These on removal were found to consist of papillomata, stuffed with the ova of the *Bilharzia*. Dr. Low, the medical superintendent of the Tropical School of Medicine, examined the fæces passed by the patient, and found ova on them, the ova having features which I will narrate presently. On rectal examination I could find nothing abnormal as far as my finger could reach, so that probably the bowel was affected higher up. With regard to the ova passed from the rectum, Zancarol teaches that the spines of ova occurring in the intestinal veins are always lateral, in contradistinction to the terminal portion of those of the ova occurring in the vesical veins. This, however, is not the invariable rule. In addition to this case, Lieutenant Lelean, R.A.M.C., has told us, in his admirable paper read last year at the Medical Society, that he has found both rectal ova with terminal spines and vesical ova with lateral.

Before proceeding to the treatment of this case a few words may be said with regard to diagnosis. The present case was originally suspected to be one of stone, and also was treated for syphilis. Now as regards the diagnosis from stone in the bladder, in both at first the hæmaturia occurs at the end of the act of micturition, this being due in the case of bilharzia to the increasing tension in the cysts as the ova reach maturity, until it is so great that rupture occurs by the contraction of the muscular coat, the blood flowing only at the end of micturition, when the muscular coat has its greatest purchase. But any pain present is not so much influenced by exercise; and the passage of the sound and the examination of the ova should settle the diagnosis.

With regard to the diagnosis of the growth due to bilharzia from condylomata, when the case came under my observation no suspicion of the latter arose to one's mind, the appearance being totally dissimilar, the surface being quite dry and not malodorous.

In the treatment of this disease the difficulty of doing any good is evidenced by the large number of remedies that have been proposed. Dr. Guillemard goes so far as to say that all treatment is useless. However, Lelean drew attention in his paper to the great benefit exercised by methylene blue given in four-grain doses three times a day. He found that in examining ova under the microscope they died in less than fifteen minutes after the stain had been run in under the cover-glass. Clinically, he found a marked lessening of the hæmaturia setting in at once. Thus he instances a case in which incessant hæmaturia had occurred for three months, two ounces of blood having been passed on an average daily, and in which there was a distinct diminution after two doses of the methylene blue, and an almost complete cessation after the fifth administration, lasting for two days. For the first few days after his admission my patient was placed on tabloids of supra-renal extract, but without any effect. I then began to try methylene blue; this was not given according to Lelean's method at first, as I ordered it every day, whereas I found Lelean only gives it for two days, as he finds the lessening of the hæmaturia is maintained for from five to seven days after one day's administration as a rule. The patient began taking four grains of methylene blue three times a day from January 15. About February 7 he took the drug in four-grain doses three times a day, for three days running. On the fourth day he was given no drug. On the fifth day he was given ten grains of benzoic acid, as recommended by Lelean, to diminish any irritability of the bladder present. Then on the sixth day the cycle was recommenced. The result, however, has been disappointing; there has been no appreciable difference in the amount of hæmaturia up to the present time. To-day I have given male fern, as recommended by Milton, of Cairo, but would be glad to hear of any suggestion for treatment from any member of this Society who has had experience of this affection.

Finally with regard to the mode of infection: there are, of course, two theories—one that infection occurs through the drinking-water; the other that it occurs whilst bathing. My patient, on being questioned, states that he frequently bathed in the pools and rivers of South Africa, and especially so when in the Nelspruit district; as to whether this had anything to do with his infection, however, I would not like to express an opinion.

XXII.—*Acromegaly, and Operation for Goitre.* By H. A. Lediard. *Read March* 27, 1903.

Mrs. M. B., æt. 43, came under observation June 11, 1902, having been sent by Dr. T. Mill, of Keswick, for operation upon an enlarged thyroid gland.

Her father died of consumption, and her mother of cancer of the uterus. Of her two sisters, one died of rheumatic fever and the other is living. Patient was born at Brampton —a goitre district, nine miles from Carlisle,—and has not lived out of Cumberland. She has been married, and nine children were born; eight of them were healthy-looking, but died in birth, her labours being difficult, instrumental, and associated with flooding. The ninth child was born alive, and is now twelve years old; another, born two years later, died at birth. On two previous occasions the patient has been under treatment in the Cumberland Infirmary. The first time was in 1887, when she complained of headache, which had existed for four years, commencing after her last confinement; the pain frequently prevented sleep, and resembled a feeling of weight and oppression in the head. The urine was saccharine and scanty, and thirst was present. A small goitre was noticed. The skull in circumference was twenty-three inches, and her sister said that the face had altered much during the past two years. The tongue was noticed to be large and flabby. It was further noted that the headache was severe, and catamenia absent. About a year later Mrs. B. was again in the Cumberland Infirmary on account of pains in the head and eyes of five years' duration, dating from the last confinement five years previously. The patient now stated that she had had to get two larger sizes of boots the last few years; her hands had been increasing for some years; her nose had altered in size; her features had swelled so that her old friends failed to recognise her. Other symptoms noted were faintness, weakness, and dyspnœa on exertion. The neck was again noticed to be thick, the lips thick, and the tongue large. All teeth were lost or had been extracted. The neck round the goitre measured 14¼ inches. The diagnosis of acromegaly had been made—this was in 1898,—and when she appeared

for the third time in 1902 the diagnosis of acromegaly was striking at a glance. The case thus presents a two-sided interest; and I will first take the story of the operation, and deal with the main features of the acromegaly afterwards. Mrs. B.'s neck had now increased to sixteen inches, and a well-marked enlargement of the thyroid was seen on both sides of the neck, especially the left; the girth increase was about two inches since 1898, or five years previously. There was now pressure on the trachea, causing dyspnœa and stridor, and it was on this account that Dr. Mill sent her to hospital for treatment.

Operation.—On June 16 the left half of the goitre was removed through a curved incision made low down in the neck. The goitre was found to press upon the trachea and œsophagus, and the lower portion had to be raised with the fingers from below the level of the collar-bone. The operation was rather more troublesome and anxious than usual, and the loss of blood was free, from the bleeding of deep veins in the root of the neck. The patient did not, however, suffer from shock, and her nervous system was undisturbed. The highest point of temperature noted was 99·2°, and the highest pulse-rate 102; whilst the healing power was quite up to the average.

The photograph marked A shows the neck prior to operation; the one marked B shows the neck eight months later. The effect of the operation, in addition to relief to breathing and swallowing, has been to reduce the size of the right or untouched side of the thyroid. This diminution is not unusual after goitre operations.

I have placed on the table the portion of the goitre removed, as well as three microscopic slides made in Professor Greenfield's laboratory.

Roughly speaking, the tumour was of old standing, and showed fibrous and cretaceous changes with cysts and blood-cysts amongst normal portions of tissue.

I now pass on to note the chief features of the acromegalous disease.

Mrs. B.'s headache and amenorrhœa have dated from her last confinement, twelve years ago, and this I take to be the commencement of the disease. In addition to symptoms already mentioned, patient complained of pains in or behind the eyeballs, and on one occasion the pain in the tongue and the feeling of increase in size was such that she consulted a doctor, fearing cancer of that organ. Household work

became arduous, difficulty being experienced in stooping or rising from the kneeling position. She easily got spent, perspired, and had a crampy stiffness in her legs. Mental failure has been evident from a lack of interest in things and a heavy feeling. A change of residence to Wythburn about a year ago, a higher elevation, has diminished the headache and cramp, and she has been brighter and freer from headaches.

Patient always had a good appetite, and for some months, about five years ago, it was so excessive as to be remarkable.

The state on admission in June, 1902, was briefly as follows:—The face has a peculiar alabaster whiteness; her features are expressionless. She seems cheerful and intelligent, and is rarely excited. The eyeballs are a little prominent; the lips large, thick, and protruding. The tongue is thick, broad, and long. The lower jaw is elongated and pointed. The skin of the face is thick, and the ears large. There is no marked alteration in the circulatory, respiratory (except stridor from pressure of the goitre), alimentary, or nervous systems. The thoracic cage is broad and the ribs powerful. There is no post-sternal dulness. The pupillary reaction is normal but sluggish. There is no urinary disorder of any kind at present. The vagina, uterus, and external organs of generation appear to be unaltered. The sense of smell is a little dull, but otherwise normal, and the senses of taste and hearing are good. With regard to muscular power, a marked diminution of power of grasp of both hands was noted as compared with that of others of similar sex, age, and build, as tested by the dynamometer. The power of walking is also poor, being slow and wanting in vigour, due possibly, in some measure, to distension of the left knee-joint with fluid. The speech is slow and clear, but the voice deep and masculine. At the present time no atrophy of any muscle has been observed. There is very slight, if any, patellar tendon reflex. The rate of the pulse is about 80 to 90, and the heart is probably enlarged, with insufficiency of the mitral valve. There are no skin growths or deposits of pigment. There is no bitemporal hemianopsia, but the fields of vision are contracted concentrically. Visual acuity $\frac{6}{36}$, and the left disc is atrophic in its outer half, and in both the vessels are contracted.

To these notes I append a table of measurements taken, but will not trouble the Society by reading it.

There is a peculiarity to note in the larynx, where the left

PLATE IV.

Illustrating Dr. Lediard's paper on Acromegaly, and Operation for Goitre.

Fig. 1.—Shows the characteristic face and enlargement of the hands.

Fig. 2.—Shows the hand of the patient compared with a normal hand.

Fig. 1.

Fig. 2.

Bale and Danielsson, Ltd.

vocal cord is not seen to be adducted in phonation, but the right cord meets its fellow without leaving any chink. This defect may be due to pressure on the recurrent nerve prior to operation, or else due to injury to the nerve at the operation. I cannot say which, as the laryngoscope was not used prior to removal of the left half of the goitre; but the immediate effect of the operation was to improve phonation rather than the contrary.

At the operation the trachea was found deflected to the right, and somewhat flattened on the left side, at the site of the growth.

The radiographs of the hands show that the width of hand is apparently due to separation of the heads of the metacarpal bones from each other. The phalanges show, or appear to show, an unusual massiveness, and the terminal phalanges show a sheaf-like tuft at their termini; this tuft may not, however, be at all peculiar to acromegaly.

The comparative photographs show the breadth of the hands of the patient and the hands of a female of similar size and age. Assuming, then, that the diagnosis of acromegaly is correct, I draw attention to the features of the case, which are striking. The colour of the face, the large, long, and broad tongue, the size of the nose and lips, the massiveness of the ribs, the shape of the hands and the great masses of tissue over the heels, and the character of the voice. It will be understood that the operation of removing a portion of the goitre was undertaken to relieve pressure upon the trachea; and the result shows, so far as a goitre operation goes, that an acromegalous woman can stand loss of blood, can bear a somewhat severe operation, and exhibit less reaction than ordinarily met with in such operations, and show a healing power fully equal to the average.

To the notes I append a detailed report of the eye condition by Dr. F. R. Hill, of Carlisle, and a report of the microscopic examination of the goitre by Professor Greenfield, to both of whom I acknowledge my indebtedness, as well as to Dr. W. Bell, house surgeon to the Cumberland Infirmary, for measurements and notes taken.

Measurements, June, 1902.

Height, 5 ft. 5 in.
Weight, 9 st. 4 lbs.
Circumference of head, 23 in.; length of head, glabella to

external occipital protuberance, by tape, $14\frac{1}{4}$ in.; by callipers, $7\frac{5}{8}$ in.; transverse breadth, maximum, interparietal, $6\frac{5}{16}$ in.; tragus to tragus, $5\frac{1}{2}$ in.; cephalic index, $\frac{6\frac{5}{16} \times 100}{7\frac{5}{8}}$. Length of nose, under surface of septum to root of nose, $2\frac{3}{8}$ in.; breadth of nose, widest part, not compressing nostrils, $1\frac{3}{16}$ in.; tragus to tragus, over vertex, tape, 16 in. (8 each side); bizygomatic breadth of face, 5 in.; lower jaw, total length by tape, $9\frac{1}{2}$ in.

Arm (upper), elbow-joint, R. $10\frac{1}{2}$ in.; L. $10\frac{1}{2}$ in.; circumference, R. 10 in.; L. 10 in.; styloid circumference, R. $7\frac{1}{4}$ in.; L. $7\frac{1}{4}$ in.

Additional Notes, February, 1903.

	June 11, 1902.	Feb. 20, 1903.
Weight	9 st. 4 lbs.	9 st. 9 lbs.
Neck circumference (in inches)	16 +	13 +

Measurements as before recorded compared with those of other individuals of same sex and build.

	Patient. Inches.	Others. Inches.
Head (with tape) measures—		
Circumference	23	21 + or −
Length (Glab. to E. Occ. P.)	$14\frac{1}{4}$	$13\frac{1}{4}$
Tragus to tragus over vertex (with tape)	16	$14\frac{1}{2}$
Lower jaw—		
Total length (with tape) angle to angle	$9\frac{1}{2}$	$7\frac{3}{4}$
Hand—		
Styloid circumference at wrist	$7\frac{1}{4}$	$6\frac{1}{4}$
Circumference	$9\frac{1}{4}$	$7\frac{1}{2}$
Length	$7\frac{5}{8}$	$6\frac{1}{4}$
Circumference of middle finger round joint	3	$2\frac{1}{4}$
Foot—		
Length	$9\frac{1}{4}$	9
Circumference at scaphoid tubercle	$10\frac{1}{2}$	9 + or −
Malleolar circumference	10	$9\frac{1}{2}$
Chest	35 + or −	36 + or −
Dynamometer.		
Right hand	10	20
Left hand	10	19

Report on the eyes by Dr. F. R. Hill (Carlisle) in case of acromegaly.

There is slight exophthalmos, but no difficulty in closing the lids. The pupil reflexes are rather sluggish, though there

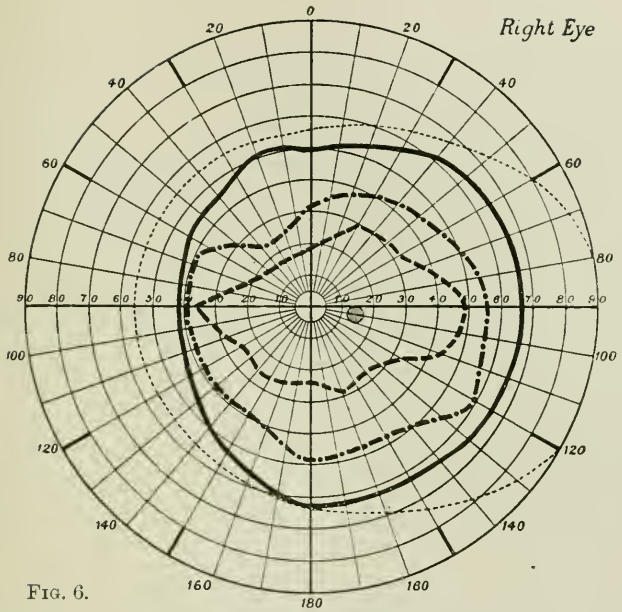

Right Eye
0
20
40
60
80
100
120
140
160
180
White Red Blue

FIG. 6.

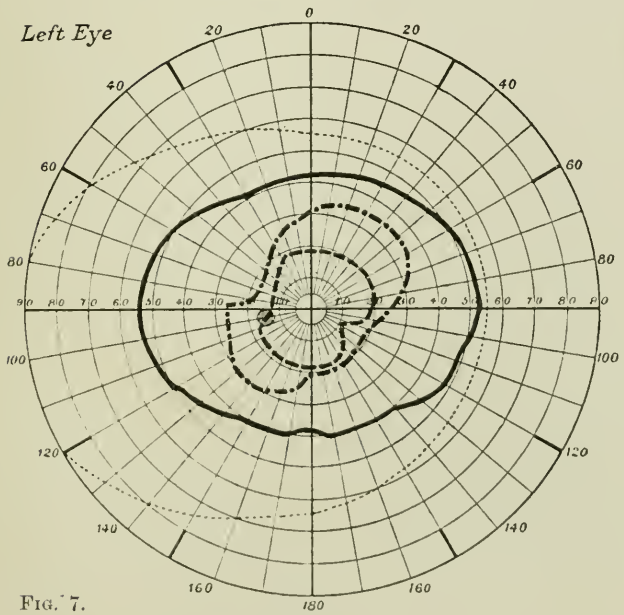

Left Eye
0
20
40
60
80
100
120
140
160
180
White Red Blue

FIG. 7.

is response to both light and accommodation. The visual acuity is $\frac{6}{36}$, but can be brought to read some of the letters at $\frac{6}{24}$ by turning head to the right and reading with left eye. The tension of both eyes is normal. The ophthalmoscope shows the right disc to be paler than normal; the left is distinctly atrophic in its outer half. The vessels are contracted in both eyes. The paler half of the left disc shows a refractive measurement of + 0, while the nasal half is − 1 D. The right eye was hypermetropic about 1 dioptre. The fields of vision show more or less concentric contraction, which is more marked when colours are used, the field in the case of green not extending beyond ten degrees in any direction.

XXIII.—*Cellulitis of the Round Ligament and Spermatic Cord, and their relation to Strangulated Hernia.*

By Edred M. Corner. *Read May* 8, 1903.

Inguinal herniæ are the most common of the herniæ of the female sex, but it is quite rare for them ever to become strangulated. The reasons for this are not quite obvious, except that the sacs are usually of no great size and have short wide necks. The inguinal canals of both sexes contain a structure—the round ligament in the female and the spermatic cord in the male—which may complicate the diagnosis of a hernia in this region. The condition of cellulitis of these structures is interesting, especially so in women, and it is to direct attention to this that these cases are brought forward.

Case 1. *Lymphadenitis and cellulitis of the round ligament simulating strangulated inguinal epiplocele.*—K. S., æt. 30, was admitted to St. Thomas's Hospital with the following history:—She had been delivered of a healthy child about one year previously. Four to five days before admission her attention had been attracted by pain to the presence of a lump in the left groin. The lump had increased in size, she had vomited irregularly, and her bowels had been open once or twice, when she had passed loose and offensive stools. There was no evidence of the previous existence of a hernia. On examination there was a tender mass over the external abdominal ring, which gave no impulse on coughing. Vaginal and rectal examinations revealed no signs of pelvic disease. The case was thought to be that of a strangulated epiplocele, with the possibility also of the existence of a partial enterocele (a Richter's hernia) as well.

Operation.—An incision was made over the swelling, and despite careful dissection no sac was found. The inguinal canal was opened up and the non-existence of the peritoneal sac proved. The round ligament was freed and found to be healthy above, whilst below it ended in the mass. It was then ligatured and the mass excised. The inguinal canal was then sutured with fine silkworm gut, healing by first intention. Patient discharged on the fourteenth day.

Examination of the excised mass showed it to consist of two parts :

(*a*) A lymphatic gland, breaking down at its centre, placed at the most prominent part, and which had done much to simulate a strangulated hernia.

(*b*) A cellulitis of the round ligament, probably of secondary origin to the lymphadenitis.

Leichtenstein and Hermann have reported a somewhat similar case in the *Monatschrift für Geburthülfe und Gynäkologie*, 1902, xv. A woman æt. 23, who had been delivered of her first child about a year previously, whilst lifting a weight felt a sudden pain and noticed a lump in the right groin. Four days afterwards she was admitted to the hospital with hiccough, vomiting, dysuria, and the bowels open. Pelvic examination revealed a hard cord passing from the broad ligament to the mass in the groin. The abdomen was opened, and the broad ligament found much contorted by old chronic cellulitis. The appendix was removed. The round ligament was found healthy. The tumour in the groin was next cut down on, and the inflamed end of the round ligament excised. Healing by first intention.

Remarks.—These two cases agree in the cellulitis of the round ligament in the groin, their general and local signs and symptoms, their mistaken diagnosis for strangulated epiploceles, the remainder of the round ligament being healthy, and that each woman had had her first child about a year previously. The chief point of difference is that Leichtenstein and Hermann's case also had chronic pelvic cellulitis, whilst mine did not. As in both cases only the distal end of the round ligament was involved, the cellulitis cannot have been due to extension of the inflammation along the ligament. Hence one must look for a local cause. In the first case the presence of a lymphatic gland was noticed, and most probably serves as the key to the explanation. The existence of a lymphatic gland along the terminal part of the round ligament is well known to gynæcological anatomists. The area which it drains is part of the fundus of the uterus and the contiguous part of the Fallopian tube. The extent to which it is connected with lymphatics of the broad ligament is quite unknown. This gland is hardly ever affected in cases of pelvic cellulitis or in cases of carcinoma of the cervix which has invaded the broad ligaments. Occasionally it becomes enlarged in cases of carcinoma of the fundus of the uterus. I would, therefore, like to suggest that the cases under con-

sideration arose primarily as a lymphadenitis due to some unhealthy condition of the mucous membrane at the fundus of the uterus, which resulted from the childbirth, and that the cellulitis of the round ligament is secondary.

The association of this affection with chronic inflammation of the broad ligament, as in Leichtenstein and Hermann's case, is possibly merely accidental, because, considering how frequent parametritis is, it would be curious that inflammation of this gland is not more common.

Case 2. *Cellulitis and thrombosis of the spermatic cord.*— The spermatic cord of a man differs from the round ligament of the female in that, apparently, it has no lymphatic gland in direct connection with it. Cellulitis of it usually results from trauma, and not from any prostatic infection. It is an uncommon condition, and must be distinguished from the superficial inflammatory condition that arises from pediculosis, etc. Whilst house surgeon in 1899 a case was admitted into the accident ward at St. Thomas's. A. B., æt. 25, had a kick in the right groin three days before admission. On examination there was a hard tender mass extending from the external abdominal ring into the upper part of the scrotum. It was tender, irreducible, gave an impulse on coughing; and there was no sickness, and the bowels were open. The overlying skin showed little bruising. After a few days in bed, with rest and hot lead lotion, the man was discharged. The mass never suppurated. The man reappeared three years later, and it was then found that there was still some thickening along his spermatic cord, and, what is extremely interesting, the testis had become smaller and softer to the touch.

It will thus be seen that these two cases illustrate in man and woman the fact that cellulitis of the spermatic cord and round ligament enters into the diagnosis of strangulated inguinal hernia, and will, I hope, serve to bring the subject more into the light than it is at present.

XXIV.—*A case of Spina Bifida (Meningomyelocele), in which the tumour made its exit through a defect at the side of the spinal column and formed an intra-abdominal cyst.* By Henry Betham Robinson, M.S. *Read May* 8, 1903.

THE case which is given in detail below is one of extreme interest and a most rare condition, for there is no clinical record to be found of a similar experience.

A female child, æt. 11 months, was sent up to me by Mr. W. J. Staddon on January 22, 1903, and was admitted into St. Thomas's Hospital. She was the first child, was born at full term, and the labour was natural. There was left talipes equino-varus with some flexion at the hip-joint; there were six toes on the left foot, due to a division of the first distally to the metatarso-phalangeal articulation. Very soon the abdomen was noticed to be fuller than natural, especially on the right side, but it had got much larger during the last two months; there were, however, no untoward symptoms present.

On admission she was a healthy-looking child, and seemed fairly bright mentally. The abdomen was very enlarged, bulging especially to the right side, without any prominence of the umbilicus. There was some impulse on straining, which did not seem more than due to increased intra-abdominal tension. The cutaneous veins were enlarged over the upper half of the right side of the abdomen. Over the right half there was marked dulness, which area seemed to have a well-defined margin at its upper part mesially, and gave a marked fluid thrill. The left side of the abdomen was resonant, and far back in the right flank there was a strip of resonance due to the ascending colon. Although on palpation it was evident there was an encysted collection of fluid, the cyst walls, as well as the abdominal wall, were quite lax until the child strained. Above, the fingers were able to dip down between the costal margin and the cyst, thus excluding a hydronephrosis. On examining the rectum the pelvis was clear, but it could

be made out bimanually that the fluid swelling came down to the pelvic brim.

The child took its food well, and there was no interference with the functions of the bladder or rectum.

As stated above, there was talipes equino-varus and some flexion of hip and knee; this condition was rather underestimated, as the limb had been manipulated in early life by a bonesetter.

There was no hydrocephalus, and there was no obvious deformity detected about the spinal column.

The diagnosis before operation seemed to point to the cyst arising either from the broad ligament and being parovarian, or from some fœtal remnant and being retro-peritoneal.

On January 26 abdominal section was performed. The belly was opened by a two-inch incision midway between the umbilicus and pubes, to the right of the mid-line through the posterior sheath of the rectus. On opening the peritoneum a bluish-coloured cyst presented, and this was tapped. A clear water-like fluid came away, more than a pint in amount. On attempting to draw out the cyst after its collapse, this was found impossible from its intimate attachment to the right side of the spine just below the transverse mesocolon. The index finger introduced inside the cyst went into a hole in the side of the spinal column. The cyst wall appeared free of nerve-structures, and its pedicle of attachment was a little more than an inch vertically. The cyst was ligatured close to the spine with silk and cut away, and the belly wall was sutured in layers.

On examining the cyst wall it was white and fibrous, with a smooth, shiny interior, and there was no appearance of any nerves in it. Its anterior pole was perfectly clear and non-vascular over a circle whose diameter was a full four inches; around this, from its site of attachment, converged five vascular buds of considerable size without any intercommunication. This condition resembled what is often seen in the posterior variety, and then due to the defective growth of mesoblast dorsally between the epidermal and neural tissues.

The fluid was colourless and limpid, with a specific gravity of 1002 and an alkaline reaction. It contained a minute trace of proteid—a body which reduced Fehling's solution, but was not a sugar—and some chlorides. No urea could be detected. The presence of the reducing body and the low specific gravity showed that the fluid was cerebro-spinal in origin.

After the operation the child lived ten days with a per-

sistent high temperature; this rose to 102·8° the night of the operation, and was never below 102°. On the 28th, at 8 P.M., it was 106·2°; on February 1, 107°; and on the 2nd, 106·6°, at 8 P.M.; after that slowly falling to 102° at the time of death on the 4th. After some initial sickness the child took its food well, and there was no alteration in the functions of the bladder or rectum.

On the 29th the head was slightly retracted, but no squint. She did not seem to respond to stimuli as actively as before, and the legs were rather flaccid. The wound was perfect and the belly quite soft.

February 1.—Anterior fontanelle fuller than before operation. The neck muscles were more rigid, but no squint. Some tetanic contraction of both hands and more flaccidity of legs, but no paralysis of bladder or rectum. From this time the child remained in about the same condition up to its death, never having any local or general fits.

On examining the spine very marked abnormalities may be seen. There is an extensive defect in the bony walls on the right side, involving the last dorsal vertebra and practically the whole of the lumbar region. At the centre of the tumour the bone defect in front is sharply defined by the lateral parts of the body centres; there are no pedicles or transverse processes, and only a small portion of the laminæ persist closely adjacent to the spinous processes. This most marked lesion seems on the level of the first lumbar vertebra. It suggests strongly that practically the whole of the lateral mass behind the position of the neuro-central suture is absent. As well as these changes laterally, the development of the bodies themselves is very irregular. Thus opposite the site of the most severe change there is almost vertical fusion of the right halves of two bodies. The vertebra above these has a very poorly formed ossific nucleus in its right half compared with its left side; while again, the third vertebra above is only a half one, showing no ossific nucleus at all in its right half. Below the site of the most marked change the next two vertebræ are altered; the nearest has a very slight development of bone on its left half, whereas the next has no bone to be seen at all in its left half. Accompanying these defects, the spine is bent laterally with a concavity to the left side.

Passing now to the contents of the spinal canal, the first thing to be noted is the marked dilatation of the central canal of the cord, as shown in section at the upper part of

PLATE V.

Illustrating Mr. Robinson's Case of Spina Bifida.

Fig. 1.—Anterior view of the spine showing the irregular formation of the vertebræ.

Fig. 2.—Lateral view of the same spine showing the cavity of the spina bifida.

Fig. 1.

Fig. 2.

Bale and Danielsson, Ltd.

the specimen. It is very evident on examination of the sac itself that the cord is involved, but it is difficult to say positively that there is any expansion of the cord at the site of the sac. That class where the expanded cord forms part of the sac wall is very exceptional, and certainly has not occurred here. The cord can be seen to the back of the sac, and from the cord can be seen nerves passing into the sac wall. The right kidney was bilobed, and lay across the upper part of the sacrum with its convexity downwards; the left kidney and other viscera were normal.

With this specimen I am kindly allowed to show one somewhat similar from the Royal College of Surgeons' Museum (No. 322), and this also figured in the Clinical Society's *Spina Bifida Report*, vol. xviii, pp. 358 and 359. Unfortunately there is no clinical record to the specimen. In this case there is a very wide defect in the lumbar region, centred in the main opposite the last lumbar vertebra, especially on the left side. There is, however, some indication at the side of the vertebra of parts developed from the lateral mass; and with the very marked defect posteriorly I should be inclined to think that the case conformed to the usual clinical type rather than to that I have the honour to bring before you this evening.

XXV.—*Cases of Angioma of Synovial Membranes and of Muscle.* By Frederic Eve, F.R.C.S. *Read May* 8, 1903.

THE following cases of angioma or nævus affecting the synovial membrane of some of the larger joints, and in some instances the adjacent muscles, may be of interest from a diagnostic point of view as well as from the fact that they have hitherto attracted little attention.

Case 1.—Florence S. was sent up to me in June, 1892, by Dr. Fraser, of Romford.

History.—Three or four years previously the patient noticed a swelling, a little bigger than a pea, just above and to the outer side of her knee. The swelling gradually increased, and at first was only occasionally painful, but had been much more so during the last month. In addition to the gradual increase, occasional variations in size were noted.

Condition on admission.—There was a swelling about the size of a hen's egg on the outer side of the thigh just above the knee. The skin over it was natural, and it was evidently deeply placed, apparently involving the upper and outer part of the *cul-de-sac* of the synovial membrane. Its outline was indistinct and consistence elastic. It was noted that on passing the hand down the thigh towards the swelling the latter seemed to get harder and to increase in size.

Operation.—I first punctured the swelling with a trocar, and blood only came away. An exploratory incision was then made and tissue opened up containing a large number of dilated vessels. On still further enlarging the incision the tumour was found to be an angioma involving the upper part of the synovial membrane and the vastus externus and crureus. It extended upwards four inches above the knee-joint and across the anterior surface of the femur to the inner side. It was removed with some difficulty owing to hæmorrhage, the knee-joint being freely opened and the affected portion of synovial membrane excised. Bleeding was arrested by ligatures, capillary oozing by the actual cautery, and the wound was plugged with iodoform gauze. The plug was removed next day and the wound closed with sutures, a drain being left in

the upper part. There was some suppuration at the upper part of the wound, but the knee-joint was unaffected, and the patient left the hospital with the wound soundly healed and a freely movable joint.

Microscopical examination.—Sections of the affected muscle showed bands of connective tissue and fat lying between and around the muscle bundles. Within these bands were very large and some smaller irregular cavernous spaces. The walls of the larger spaces were as a rule very thin, but in places a thin band of unstriped muscle could be traced. The

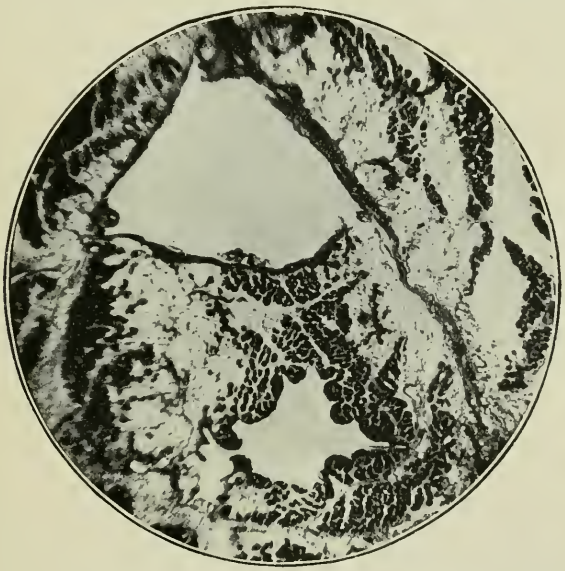


Fig. 8.—(Obj. Zeiss A.) Section of a cavernous nævus of the synovial membrane of the knee-joint. It shows freely communicating cavernous spaces separated by thin partitions of connective tissue.

walls of the smaller spaces were thicker. There were also blood-vessels, showing irregular dilatations of their lumina, the walls of which possessed a distinct layer of unstriped muscle (*see* Fig. 9).

I have since met with a precisely similar case, also in a young woman in whom an angioma involved the upper part of the synovial membrane of the knee-joint and the adjacent muscles. In this case the diagnosis was made owing to the resemblance to the case first related. I am unable to give details owing to the notes having been mislaid.

Case 2.—I saw Master D. with Dr. J. Burton in March, 1898. The left knee had been painful for four or five years without evident cause. The pain was growing worse, and he had begun to experience difficulty in moving the joint. Latterly a swelling had appeared on the inner side. He stated that the knee started in his sleep. A maternal uncle and one of his cousins had died of consumption.

Present condition.—On the inner side of the left ligamentum patellæ was a prominent swelling, which was elastic over the inner two thirds and fairly well defined over the outer third. The swelling, after use of the limb, became hot, tender, and red. The skin over it was normal. Measure-

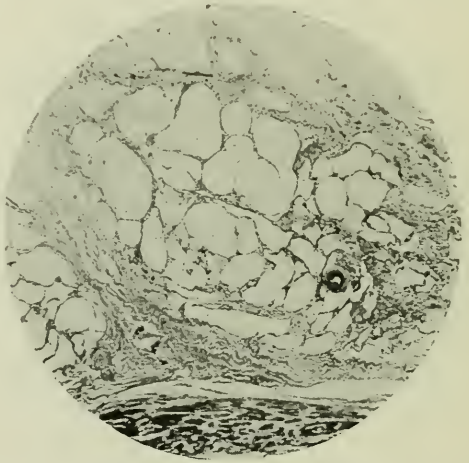


Fig. 9.—(Very low lenses.) Section of the vastus externus muscle showing large irregular cavernous spaces. Bands of connective tissue lie between and around the muscle fasciculi.

ments showed a diminution of circumference of the left thigh of one inch, and of the left calf of half an inch. The circumference of the knee-joint over the patella was unaltered, but above it the left thigh was diminished owing to the wasting of muscle. It was thought to be a case of localised tuberculosis of the synovial membrane such as I have occasionally met with; and as the function of the joint was becoming impaired I advised operation.

Operation.—A transverse incision was made extending from the inner side of the ligamentum patellæ, over the inner aspect of the joint, and following the line of the articulation. The capsule was opened and a roundish swelling of a yellow colour

exposed, presenting at first sight the appearance of caseous tubercle; it was, however, spongy in consistence. Its real nature was recognised by an incision, and it was completely removed. The capsule of the joint was united with silk and the skin sutured. Primary union took place, and movement of the joint was completely restored.

Microscopic examination.—The sections presented the typical appearance of a cavernous nævus. The blood-spaces were very large, freely communicating, and with very thin walls (*see* Fig. 7). In parts of the section there was a good deal of dense fibrous tissue.

In the two following cases a small circumscribed angioma was situated near the elbow-joint.

Case 3.—Mary B., æt. 23, was admitted to the London Hospital in October, 1898.

History.—Twelve months previously her elbow became stiff and painful, the pain being worse at night, and there was often starting of the limb at night. She could not use the arm on account of pain. Her left knee was swollen three years ago, and was treated by strapping for three months.

Present condition.—There was a swelling on the inner side of the elbow between the olecranon and the internal condyle, which also extended somewhat to the front; it was elastic, and the synovial membrane near it felt thickened. Flexion of the joint was limited, and the muscles of the arm were flabby. The tumour was acutely tender.

Operation.—An incision about two and a half inches long was made between the olecranon process and the internal condyle. The ulnar nerve was drawn aside and the capsule opened, when a mass of thickened synovial membrane was exposed. An incision revealed a few soft yellowish bodies, which proved to be phleboliths. Primary union with restoration of movement took place.

Microscopic examination.—In this case the angioma was entirely confined to the synovial membrane. The cavernous spaces were very large, with thin walls, and were widely separated by connective tissue. Many of them were filled with fibrin, which to the naked eye presented the appearance of caseous material.

Case 4.—Alfred A., æt. 24, was admitted to the London Hospital at the same time.

History.—He complained of a lump on the outer side of

the right elbow, which had existed for three months. It was slowly increasing, but caused neither pain nor limitation of movement.

Present condition.—On the outer side of the right elbow between the olecranon and external condyle was an oval elastic swelling the size of a pigeon's egg. It was smooth, well-defined, movable laterally, and not tender.

Operation.—An incision was made over the swelling in the axis of the limb, and a fatty-looking growth which bled rather freely was removed from the substance of the triceps and anconeus and the neighbouring synovial membrane. Primary union.

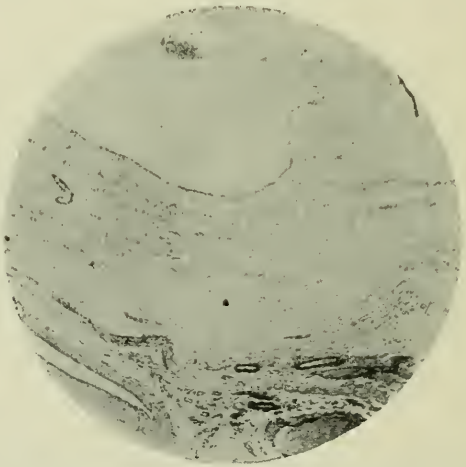


Fig. 10.—(Obj. Zeiss A.) Section of a nævus of the synovial membrane of the elbow-joint. It shows above a large vascular channel, and below some smaller blood-vessels.

Microscopical examination.—The chief part of the mass was composed of fat, but there were also bands of connective tissue containing numerous small and some larger venous channels; in other parts were much larger vascular channels with ill-developed walls. At one point there were bundles of degenerate muscle-fibre separated by bands of connective tissue containing numerous capillary blood-vessels.

Remarks.—The feature of chief interest arising in connection with these cases is the question of diagnosis. All occurred between the ages of ten and twenty-five, when tuberculosis of the joints is common; some of them undoubt-

edly complained of symptoms considered to be characteristic of tuberculosis, such as pain increased by movement, and two complained of starting of the limb at night. Tenderness and limitation of movement were marked in three cases, and in two wasting of muscle was noticed. In some instances the swelling is described as being soft and elastic—in other words "pulpy." We cannot be surprised, therefore, that in the majority of cases a diagnosis of localised tuberculosis of the synovial membrane was made; while in the last case lipoma was suggested, and, in fact, the tumour consisted chiefly of fat.

Pulsation was not observed, but in the first case it was noted that the swelling increased when pressure was made above it. Pressure upon the venous trunks above the tumour might perhaps have proved a diagnostic sign in some of the other cases had it been tried. Emptying of the tumour by pressure was not apparent. Possibly in Case 3 the symptoms were exaggerated by a neurotic tendency, just as they may be in fibro-adenomata of the breast of young women.

The histories pointed to a somewhat rapid increase of the tumour in Cases 1 and 2; and in the former the microscopic appearances suggested a progressive infiltration of muscle by fibro-vascular tissue. In Cases 2 and 3 the angioma was entirely confined to synovial membranes.

I mentioned Case 1 in a discussion on a case of nævus of the peritoneum recorded by Mr. Arbuthnot Lane.* A careful search through literature has failed to find any additional cases of angioma of the synovial membrane of joints. They cannot be very rare, as I have met with the four cases recorded and seen two others. Probably they would have remained unrecognised but for the advances in surgery which have enabled us to open joints with safety.

In an admirable monograph entitled "Remarks on some Cases of Vascular Tumour seated in Muscle," † Mr. Campbell de Morgan describes two cases of vascular tumour, definitely circumscribed and encapsuled, situated respectively in the gastrocnemius below the popliteal space, and on the front of the thigh about its middle, within the substance of the rectus. He gives a complete collection of similar cases published up to date, quoting examples of vascular tumour involving the posterior muscles of the neck, supinator longus, deltoid, rectus

* *Clinical Soc. Trans.*, vol. xxv, p. 5, 1893.

† *British and Foreign Medico-Chirurgical Review*, vol. xxxiii, 1864, p. 187.

femoris, inferior and external part of the thigh ; and also one on the side of the chest which involved the latissimus dorsi, serratus magnus, and intercostal muscles. Some of them were well-defined and encapsuled, others diffuse. In one case recorded by Lister there was distinct pulsation ; in some the blood could be expressed from the tumour, while others were mistaken for lipomata until they were incised.

XXVI.—*A case of Hair-ball in the Stomach.* By Charles A. Morton. *Read May* 8, 1903.

H. T., æt. 18, was admitted into hospital, under my care, at the beginning of December, 1902, with a large and very hard abdominal swelling, which had been noticed for some months only. Four months before admission she began to vomit, but the vomiting did not occur after food, as one would expect if the stomach was already occupied by a large mass of hair, but in the night, and before meals. Indeed, she would take a crust to bed with her to eat in the night if the sickness came on. At the time when the vomiting began she said she suffered from almost constant pain across the epigastrium, but both the pain and the vomiting had almost ceased at the time she was admitted. For several years there had been occasional pain in the right side of the abdomen.

After the operation we ascertained from her mother that eighteen months ago she had been seen to pull out her hair and eat it, and at the age of twelve years she was known to eat pieces of cotton. We could get very little information about this from the girl herself, but she was not of unsound mind.

The position of the swelling is shown in the diagram, and, as I have already stated, it was very hard. Its range of movement was remarkable, for it could be readily pushed down to the left anterior superior spine, and it always spontaneously returned to its original position. It was also very resonant. From its mobility and resonance it seemed to me more like an omental swelling than any other. I had never seen a tuberculous mass in the omentum as large, or as heavy, or as movable, but it seemed possible it might turn out to be of this nature. The resonance was due to the stomach note over the mass of hair.

It is curious what various tumours, masses of hair in the stomach have simulated. In Mr. Paul Swain's case * it was also thought to be an omental growth; in Mr. Knowsley Thornton's † it was first thought to be a fæcal concretion in

* *Lancet*, 1895, vol. i, p. 1581.
† *Ibid.*, 1886, vol. i, January 9.

the colon; and, indeed, my own case had been under treatment in another hospital for this condition before she came under my care. In Mr. Donald Day's case * cystic disease of the spleen had been diagnosed before operating. Dr. Fenwick, in his recently published paper on hair-balls in the stomach,† says that most cases are diagnosed as cancerous growths if gastric symptoms are present. The early age at which these hair-balls are usually found would be, as he points out, suggestive that the swelling was not a malignant growth of the stomach. The remarkable range of mobility is also a striking feature in these cases.

In the case which Mr. Paton brought before this Society lately ‡ in a child, the known habit of eating hair suggested the nature of the mass; and in Mr. Knowsley Thornton's case, although at first a fæcal mass in the colon was diagnosed, yet later the passage of hair with the fæces suggested the true nature of the swelling, though even then it was thought to be in the colon rather than the stomach.

On December 8 I removed the mass. It was necessary to make a 4-inch incision in the stomach. This organ was much dilated, but only one small ulcer was seen in it. There was some hæmatemesis during the first forty-eight hours after the operation, but after that she had no further symptoms, and made a good recovery.

The mass when removed from the stomach was covered with green mucus, and impregnated with some solid material which could be washed out, but which made its weight as much as 1 lb. 12 oz. The size of the hair-ball was 6 by 6½ inches, and 2½ inches in thickness. I was able easily to extract it through the 4-inch incision, as the stomach wall stretched.

* *Brit. Med. Journ.*, 1902, vol. ii, p. 1899.
† *Ibid.*, 1902, vol. ii, p. 1696.
‡ *Trans. Clin. Soc.*, vol. xxxv.

XXVII.—*A case of Ileo-cæcal Invagination produced by a Meckel's Diverticulum, which was likewise invaginated; operation; recovery.* By T. P. zum Busch. *Read May* 22, 1903.

S. K., æt. 21, consulted me on November 21, 1902, and gave the following history:—For more than a year he had suffered from dull pain in the abdomen, chiefly round the umbilicus, and from retching after meals, and from occasional attacks of diarrhœa, alternating with days or even weeks of constipation. Blood or mucus had never been noticed in the fæces. The patient, an acrobat, had consulted many specialists in different parts of Europe, but had never had time to remain under clinical observation. Examination of the patient, who was very muscular, showed nothing abnormal, except that the muscles of the right side of the abdomen seemed more prominent and rigid. This, however, was explained by the patient as being due to his occupation. There was no tenderness or swelling, nor did careful examination reveal a hernia or anything abnormal in the rectum. Not being able to make a diagnosis, I advised the patient to come into the German Hospital for observation, which he promised to do at once. He was, however, not able to leave the stage the same day, but had to act his part on two more evenings. The work, which he acknowledged to have performed only with great difficulty and agonising pain, consisted in serving as the base of a pyramid of ten persons, which he balanced on his feet. The strain on the abdomen was all the greater as these persons did not remain quiet, but turned somersaults in the air, always alighting on his outstretched feet. Shortly after the performance on Saturday night he became very sick, and vomited a great deal. He felt a great inclination to defæcate, and passed, as he thought, several liquid motions during the night and the following day. Sunday evening (the 23rd) he was admitted into the German Hospital, where I saw him early the next morning. The patient now seemed very different from the man I had seen three days previously. His expression was pinched, and he was greatly collapsed. The

pulse was small, slightly irregular, 120 beats in the minute. He had retained no food, but had vomited large quantities of dark, bile-stained fluid. There was a great deal of tenesmus; the so-called motions consisted almost entirely of blood. The segments of the right rectus abdominis formed big lumps, whilst the left rectus was quite flaccid. A swelling the size of a child's head, and dull on percussion, filled the ileo-cæcal region. There was no doubt that we had to do with an invagination, probably into the cæcum; in view of the long-standing abdominal trouble, the further diagnosis of an intestinal tumour, perhaps of a polypoid nature, was made, and this tumour was thought of as the probable cause of the invagination.

The patient being willing, he was at once anæsthetised and prepared for operation. As soon as the abdomen was opened by an oblique incision over the cæcum, a large mass presented itself, which, on closer examination, was found to be part of the ileum invaginated into the cæcum. It was exceedingly difficult to disinvaginate this mass, and at one time it seemed almost impossible. Gradually, however, the invaginated part was freed, and as the last piece came out a considerable quantity of dark-stained fluid escaped—a fluid which looked exactly like the one that is seen in a strangulated hernia.

The invaginated intestine, after being freed, was found to contain in its upper part a hard globular swelling, which occluded the lumen almost completely. It was further found that the whole length of the invaginated gut was gangrenous, and would have to be removed. In spite of the operation having lasted already fifty minutes, I decided to excise the gangrenous part and unite the stumps at once, instead of exposing the patient to all the dangers and drawbacks of an artificial anus in the ileum. The intestine was therefore clamped and resected close to the cæcum, and again about three feet higher up. Both ends were invaginated and closed by a double row of continuous sutures. The parts were then joined by a large lateral anastomosis, no bobbin or other artificial help being used. The lateral anastomosis was preferred to an end-to-end one, as the intestinal walls were very œdematous, and it seemed safer to bring together as large surfaces as possible. A Mikulicz tampon was brought down to the anastomosed intestine, and the greater part of the abdominal wound was closed. The patient, after an operation of $1\frac{3}{4}$ hours' duration, was very collapsed, but rallied well on subcutaneous injections of normal saline fluid—camphor and

ether. The sickness ceased at once; the bowels acted spontaneously thirty-six hours after the operation; recovery was uneventful, except that a small abscess in the line of suture necessitated the removal of a few stitches. The patient was allowed to get up on December 26, and left the hospital on January 7. He has been travelling with his troupe and practising a new trick, but an abdominal hernia in the scar necessitated a second operation, which was done yesterday.

The portion of gut removed constituted the last three feet of ileum. In its upper part it was nearly occluded by a rounded polypoid mass, which on closer inspection proved to be a Meckel's diverticulum, which had become invaginated into the ileum. This invagination of the diverticulum had evidently existed for some time, and had probably been caused by a subserous lipoma the size of a hazel-nut, which was situated at the apex of the diverticulum. The obstruction caused by this primary invagination, together with the enormous strain thrown on the abdomen by the patient's occupation, had during the last few days caused the secondary invagination of the lower three feet of ileum into the cæcum.

In looking through the literature on the subject we find that ileus has not infrequently been caused by a Meckel's diverticulum. In the majority of the 195 cases which Hilgenreiner (1) has collected in a recent paper, the condition found at operation or necropsy showed that the diverticulum had become adherent to some part of the abdominal wall or to a piece of intestine; it had in this way formed a loop or bridge through which a piece of gut had passed and become strangulated. In other cases adhesions between the diverticulum and the intestine had led to kinking of the latter or to volvulus and similar conditions. Although a consideration of these different forms of intestinal obstruction is very interesting, it would lead us too far to-night, and we shall, therefore, only consider those cases in which an invaginated Meckel's diverticulum has given rise to the occurrence of intestinal obstruction.

Of these cases we find fifteen in the literature on the subject. In three cases the diverticulum alone had become invaginated and led to signs of obstruction. Two of these cases were treated by operation, one without; all three died. In twelve cases an invaginated diverticulum had given rise to a secondary invagination of intestine; in six of these twelve an invagination of ileum into ileum, in five of ileum into cæcum, and in one of ileum into colon were described. Only

in three of the whole fifteen cases a probable cause for the invagination of the diverticulum could be given. In two cases a small accessory pancreas, in one case a small subserous lipoma, were situated at the apex of the diverticulum, and by their weight they probably caused the invagination. In most cases (twelve out of fifteen) the primary invagination of a Meckel's diverticulum led, as already mentioned, to a secondary intestinal invagination. Of these twelve cases four were not treated by operation, and three of them died. One, however, recovered spontaneously. This very interesting case, which has been reported by Hirschsprung (2), occurred in a boy æt. 7. After complete obstruction, which lasted for eleven days, a piece of intestine 40 cm. long, with an adherent invaginated diverticulum, was passed *per rectum*, and the child recovered. The other eight cases were treated by operation. In three of them disinvagination was easy, and the intussuscepted part was in such a good condition that an excision seemed unnecessary. In spite, however, of the comparatively small operation, only one case recovered, whilst two died.

In five cases the intussuscepted part was so damaged that it had to be excised. Of these five cases, in which an enterectomy had to be performed after the disinvagination, only one case recovered, whilst four succumbed to the operation.

The successful case was published by Brunner (3). The patient was a boy æt. 4, and 20 cm. of ileum were removed, together with a diverticulum. The latter bore at its apex an accessory pancreas; and it is not quite certain whether the organ found was really a Meckel's diverticulum, as Nauwerck (4) has described a case in which two diverticula were found in the ileum, one being a regular Meckel's diverticulum; the other, apparently very similar in structure, was situated more centrally, and bore an accessory pancreas at its apex.

If we accept Brunner's case as one of Meckel's diverticulum, it is the only published case in which an intestinal obstruction caused through an invaginated Meckel's diverticulum and a secondary intestinal invagination has recovered after excision. The case reported by me would then be the second successful case. In Brunner's case, however, only 20 cm. of ileum were resected, whilst in my case over 100 cm. had to be removed.

References.

1. Hilgenreiner, "Darmverschluss durch das Meckel'sche Divertikel," *Beiträge zur klinischen Chirurgie,* Band xxxiii, Heft 3.
2. Hirschsprung, cit. by Hofman, *Beiträge zur Pathologie des Divert. Meckelii,* Inaugural Dissert., Kiel, 1897.
3. Brunner, "Darmverschluss durch Meckel's Divertikel," *Beiträge zur klinischen Chirurgie,* Band xxv, p. 351.
4. Nauwerck, quoted by Brunner.

XXVIII.—*Sequel to a case shown at the Society in* 1901 *as Congenital Morbus Cordis; Diffuse Endarteritis of the Pulmonary Arterial System.* By Percy Kidd, M.D. *Read May* 22, 1903.

Susan G., æt. 21, was shown on February 22, 1901, suffering from dyspnœa and attacks of cardiac pain, the latter dating from childhood. There was no history of rheumatism. The girl, who was of very small size for her age, was slightly cyanosed, but there was no clubbing of fingers.

The physical signs indicated enlargement of the heart, mainly of the right side. The apex-beat was in the fifth space in the nipple line, and there was a strong diffused impulse in the third, fourth, and fifth spaces, reaching to the right border of the sternum.

The cardiac dulness extended upwards to the third rib in the left parasternal line, outwards to the nipple line, and inwards to the right border of the sternum. On auscultation a loud, short, shock sound was heard over the whole precordia, resembling the first sound of mitral stenosis. This proved to be the second sound, and its point of maximum intensity was in the pulmonary area, in which position a faint, short diastolic murmur could occasionally be heard following the second sound. The first sound was weak in all areas, but was unaccompanied by any murmur. The lungs and other viscera appeared to be healthy.

From the history, symptoms, and appearance of the patient, together with the signs of hypertrophy of the right ventricle, it seemed clear that she was suffering from congenital disease of the heart. This view was accepted by most of the members of the Society who examined the patient. Little or no change occurred in her general condition for the next six months, but early in August, 1901, she began to suffer from nausea, vomiting, and dyspnœa; the liver was found to be enlarged and tender, and the ankles became œdematous. On August 17 the patient had a sudden attack of dyspnœa, with marked lividity, and death ensued in a few minutes.

Abstract of the post-mortem *examination by Dr. Horton-Smith.*

Heart, seventeen ounces; right side, both auricle and ventricle, obviously dilated and hypertrophied, this being most markedly the case in the ventricle. Left ventricle and auricle of natural size. Tricuspid orifice somewhat enlarged, easily admitting three fingers and a cone of $4\frac{3}{4}$ inches circumference. The pulmonary valves showed on each cusp at their centres small patches which suggested recent endocardial vegetations. With this exception the valves on both sides of the heart were all natural. Neither aortic nor pulmonary orifices allowed regurgitation. Hence there seems nothing in the heart to account for the hypertrophy and dilatation of the right side. The muscular tissue of the heart is healthy; coronary arteries natural. Aorta showed very slight atheroma above the valves. Pulmonary artery distinctly atheromatous in its trunk and great branches, while the divisions of the artery which ramified on the lungs were found to show atheroma of quite a high degree. The vessels which ran in every lobe were marked with numerous yellow patches scattered throughout their walls, which in some places were fused into large atheromatous tracts. No calcareous change. The arteries stood out much more clearly than usual, owing to their atheromatous condition when the lung had been incised. Lung tissue engorged, but otherwise healthy. Left pleura adherent throughout. Other organs normal.

Report on the Microscopical Appearance of the Pulmonary Arteries, by F. C. Shrubsall, M.D.

Sections have been cut through the main divisions of the pulmonary artery and through its finer ramifications in the lungs. Throughout a condition of chronic endarteritis was found, the tunica intima being greatly thickened everywhere, but more especially so in the areas appearing to the naked eye as raised yellow patches. The capillaries in the lung were greatly engorged, but their walls, although perhaps a little thickened, did not present the marked changes seen in the arteries.

Sections through the smaller vessels showed a general thickening of the intima, in places greatly exaggerated.

The subendothelial layer presented a cellular proliferation, with a great profusion of round and ovoid cells. On staining by von Giessen's method it appeared that adjacent to this was a marked increase in the fibrous tissue interspersed with round and stellate cells. The lung tissue around was normal.

The larger vessels presented somewhat similar appearances, but here there was much more fibrous tissue in the intima, forming an interlacing network of strands bounding lacunæ, which contained poorly staining granular material, probably coagulated lymph. There was less cell-proliferation evident, and the process appeared to have passed on to actual fibrosis, affecting in some degree all the coats of the artery. In no place was there evidence of definite fatty or calcareous degeneration, or of much round-cell infiltration, the condition appearing to be one of a simple chronic endarteritis without definite atheromatous change.

The results of the autopsy occasioned much surprise, and I thought it incumbent on me to bring the facts before the Society, as illustrating a most unusual case. There can be no question that the hypertrophy of the right ventricle must be attributed to the wide-spread endarteritis of the pulmonary arterial system. It is very difficult to account for this condition in the absence of any chronic disease of the lungs or mitral valve. The history of cardiac symptoms dating from childhood suggests that the arterial disease originated in intra-uterine life.

A very similar case is reported by Romberg ("Ueber Sclerose der Lungenarterien," 'Arch. f. kl. Med.,' 1891–2).

This case also was regarded during life as one of congenital morbus cordis. The pulmonary artery throughout showed a condition of arterio-sclerosis, and the chief branches were dilated. The aorta, mitral valve, and the lungs were healthy.

XXIX.—*Further History of a case of unusually Great Dilatation of the Heart.* By J. Porter Parkinson, M.D. *Read May* 22, 1903.

THIS patient was shown at the Clinical Society's meeting in January, 1901, and since then has been almost constantly under observation; and I have been encouraged to read the further notes of the case by the interest it excited previously, and by my being able to show the cardiac condition.

The patient was a man æt. 22, with a rheumatic history; he was undersized and thin, with slight cyanosis; pulse 90, small volume, and irregular in size and rhythm. Slight enlargement and systolic pulsation of jugular veins. The cardiac dulness was shown in the diagram, and presystolic and systolic murmurs were heard at the points indicated in it. The liver was enlarged and showed systolic pulsation, and the other organs showed signs of congestion, but there was no dropsy, either of the limbs or abdomen.

Such is a brief sketch of his condition in January, 1901, and there was but little change till anasarca and ascites came on in July, 1902. Sixty-four ounces of ascitic fluid were removed on July 15, and 74 oz. on July 22, after which the fluid did not re-accumulate to any marked degree; and the anasarca diminished so that he went out of the hospital in September, to be readmitted a month later with dropsy of the legs and abdomen as before, which, as it increased, necessitated tapping, and 90 oz. were removed on November 12, and 50 oz. on November 16. The dropsy, however, again reappeared, and involved the pleuræ, and he finally died on December 14, 1902. Throughout the case, till the last few months, the most marked feature was the absence of cardiac symptoms, such as orthopnœa, etc., or any visible distress.

At the *post-mortem* examination—conducted by Dr. Byrne, Medical Registrar and Pathologist,—on opening the thorax almost the whole space of the front of the chest below the second costal cartilages was seen to be occupied by the distended pericardium, only a small portion of lung being visible above, as is seen in the diagram drawn on the spot.

The parietal pericardium was so firmly adherent to the visceral layer that it had to be removed in pieces before the various chambers of the heart could be made out; and it was seen that both auricles were greatly distended, and the right ventricle was also much enlarged, forming the greater part of the heart as seen from the front, and also the apex. Thirteen ounces of blood-clot were removed from the left auricle, and the right auricle was even more capacious; the auricular walls were thin, and appeared to be almost devoid of muscular tissue. The ventricles were somewhat dilated, especially on the right side; the thickness of their walls was approximately equal, amounting to about five eighths of an inch.

The mitral valve was much stenosed, forming a slit about 3 cm. long and 1 cm. wide, not admitting the tip of the finger; the valves and chordæ tendineæ were thickened and contracted. The tricuspid orifice was slightly stenosed, and the valves were obviously incompetent, being thickened and sclerosed. The other valves were normal. The weight of the empty heart was 30 oz. Since the removal of the heart it has shrunk very considerably, and hence its present appearance gives no idea of its original size.

Both lungs were collapsed at their bases, and contained scattered patches of broncho-pneumonia. There was some fluid in both pleuræ, and the abdomen contained several pints of free fluid. The liver was much enlarged, weighing 64 oz.; it was tough, and typically nutmeg in appearance. The spleen, kidneys, and pancreas and other abdominal organs showed evidences of chronic congestion.

I show the specimen as a contribution towards a subject which has been discussed during the past two years in papers by Sir Isambard Owen and Dr. Ewart, and especially as my case has been alluded to by Dr. Ewart as a possible example of "a pulsation over the auricular district of the precordium produced by immense enlargement of the left auricle" (*Clin. Soc. Trans.*, vol. xxxv, p. 153).

It seems from the *post-mortem* appearances that the systolic pulsation felt during life just below the right nipple was produced by the right auricle, which lay immediately beneath; and, judging from the very small amount of muscular tissue in either auricle, that the impulse could not have been produced by their contraction, but was due rather to the regurgitation of blood from the hypertrophied ventricles during their contraction. Owing to the firm adherence and thickening of the pericardium the actual position of

each auricle was not completely made out before the removal of the heart, but it appeared that the dilated left auricle bulged backwards and to the right, and did not present to any great extent on the anterior aspect of the heart.

This case seems, to some degree, comparable with those recorded by Sir I. Owen and Dr. Ewart, but the enlargement of the left auricle appears by no means so great, and, indeed, is less in capacity than the right auricle. Again, my case differs in being associated with and probably due to mitral stenosis, and the tricuspid valve is also thickened and deformed, neither of which features was present in the cases referred to. On the other hand, all the clinical signs of horizontal dilatation of the left auricle mentioned by Dr. Ewart and Sir I. Owen were present in my case; and especially noted was the singular absence of cardiac distress, dyspnœa, orthopnœa, and dropsy, for it was only during the last few weeks of life that ascites appeared.

CLINICAL CASES

EXHIBITED.

*** Published in accordance with the regulation relating to the exhibition of patients at the meetings of the Society, viz. that each case shall be accompanied by a brief written description of the points it illustrates; such description to be published or not in the *Transactions* as the Council may direct.

I.—*Pressure Paraplegia successfully treated by Resection of the Head of a Rib and the adjacent Transverse Process.* By J. Jackson Clarke. *Exhibited October* 24, 1902.

Eliz. R., æt. 4, admitted to the North-west London Hospital May 11, 1901. A weakly, thin child, with an angular projection in the upper dorsal region of the spine and loss of power in the lower extremities. She could only stand up for a few seconds by herself. Treated by complete rest in bed and fixation of the spine, abundant food, air, and cod-liver oil for eight months; the paraplegia had, with slight fluctuations, steadily got worse, and was accompanied by marked wasting of the limbs. A skiagram was taken, and it showed a round area of darkness with its centre opposite the fifth dorsal vertebra. The dark area was interpreted as being due to tuberculous and inflammatory infiltrations, and the above-named operation (described, under the designation of "costo-transversectomy," by Ménard in 1894) was decided on, and carried out on January 15, 1902.

The right transverse process of the fifth dorsal vertebra and the neck and head of the corresponding rib were removed, and a sharp spoon being passed to the front of the spine, a little firm caseous material was removed and some iodoform-glycerine-carbolic emulsion injected. The immediate result of the operation was that the previous limp and powerless condition of the limbs was replaced by a slight degree of spasticity. This passed off in a few days, and the condition was again the same as had been observed before the operation. Seven days after the first operation the track leading to the pre-vertebral tuberculous area was again opened up under an anæsthetic and scraped; this time a tube was inserted so that a little of the emulsion could be injected daily. Again the slight spastic condition showed itself, but this time the voluntary movements improved also, the child at first being afraid to move her limbs owing to the excessive and painful spastic response. The spastic condition soon began to pass away, and the voluntary power steadily increased. The patient has now been running about in a totally normal way for some months. The deformity of the spine has been lessened by the mode of fixation employed.

II.—*Congenital Shortening of one Rectus Abdominis Muscle.* By J. Jackson Clarke. *Exhibited October 24, 1902.*

A GIRL, A. B., æt. 10, was shown before the Society last session on account of a congenital shrinkage of the right rectus abdominis muscle, the condition of which was then thought to be similar to that of the sterno-mastoid muscle in congenital wry-neck. This surmise has since been proved to be correct. The muscle was divided by open operation and the previously kyphotic spine was over-extended, and during this proceeding the divided ends of the altered (fibrosed) muscle separated for three inches within the sheath. The gain in mobility of the spine and in respiratory power has been marked.

III.—*Three cases of Family Progressive Spinal Muscular Atrophy (Werdnig-Hoffmann type).** By Frederick E. Batten, M.D. *Exhibited October* 24, 1902.

CASE 1.—D. W., a boy æt. 7, was a healthy baby, and walked at twelve months. From that time onward progressive wasting of the thighs and weakness of the legs has been noticed. The boy talked at eighteen months old, and can read and write. He has had no pain, and the onset of the weakness has been gradual. There has never been any trouble with micturition or defæcation.

Family history.—The mother and father are both healthy, and are not in any way blood relations. Both mother and father have numerous brothers and sisters, none of whom, nor their children, present any weakness. The family consists of seven children, of whom this boy is the fifth. There are four boys and three girls :

1. Boy, æt. 18, healthy. Suckled five months.

2. Boy, died when 10 years old; had been paralysed since birth. Suckled.

3. Boy, died when 4 years old; weakness came on when 14 months old. Suckled.

Premature birth at the eighth month.

4. Girl, died when 5 years old; paralysed in the legs. (Notes of this case, seen in 1895, appended.) Suckled nine months.

5. Boy, æt. 7. (Patient shown, Case 1.) Suckled.

6. Girl, æt. 6, healthy. Not suckled.

7. Girl, æt. 1½. (Patient shown, Case 2.) Suckled eleven months.

The boy is pale and thin, he walks with considerable difficulty, and there is marked lordosis. The gait is waddling. When placed on the ground and asked to rise, he does so by first sitting up by flexion of the abdominal muscles in the ordinary way, and then drawing his legs up under himself and assisting their extension with the hands.

All movements of the face are well performed—he can whistle and protrude the tongue well. All the ocular move-

* Two of the cases were shown before the Society; the third was seen seven years ago, and is here recorded.

ments are natural. All movements of the arms can be performed, but the scapulæ tend to wing when the arms are held out in front. The whole of the trapezius muscle is small, and the muscles about the upper arm are weaker than those of the forearm or hands. The pectoralis major is well developed. The muscles of the back are weak. The muscles of the abdomen are good.

There is marked weakness of the flexors and extensors of the thighs, weakness of the flexors and extensors of the legs, but the muscles below the knee are not so weak as those of the thigh. There is weakness of the dorsi-flexors of the feet. There is no hypertrophy of any muscle; there is wasting of the thigh muscles; there is no alteration in sensation.

Reflexes.—The knee-jerks are absent. The plantar reflex gives a flexor response on both sides.

Electrical reaction.—There is a diminished reaction to faradism in the affected muscles; the unaffected muscles react well.

Case 2.—M. W., æt. 1½, sister of above.

Child was strong and healthy at six months of age; she could stand and would "walk up" her mother. When twelve months old the weakness and wasting of the legs was first noticed, and now she is quite unable to stand, and when asked to move her legs she does so by grasping the thigh with her hands.

She is a perfectly intelligent child, and talks well. The face and arms are normal in every respect. The trunk muscles are weak, for although she can sit up, yet her balance is most easily upset, and there is difficulty in assuming the sitting posture again. The legs, and especially the thighs, are flabby and weak, and there is no power of flexion or extension of the thighs on the trunk, nor of the leg on the thigh. The extension and flexion of the foot and toes are well performed.

Reflexes.—The knee-jerks are absent. There is no alteration of sensation.

Case 3.—D. W., girl æt. 3 years 4 months, seen in April, 1895. She was the fourth child in the family, and died when five years old. *No autopsy.*

History.—The child had never walked, but when twelve months old had more power in the legs than she has now. She could crawl and move her legs fairly well; now she has to lift her legs up with her hands. There has been no dis-

turbance of general health, and the child has never had measles, whooping-cough, or scarlet fever.

The child is bright and intelligent, speaks with a slight lisp, but well for a child of her age. All movements of the head and neck are well performed. The arms are thin, but there is no wasting of any particular group of muscles, and all movements are well performed. The legs are thin and wasted; there is, however, no movement which the child cannot perform while lying in bed. She is quite unable to support her weight on the legs.

When placed on her back she is quite unable to raise herself, and even with the aid of the sides of the cot she cannot get up. When on the floor the child rotates herself on to her face, flexes the thighs on to the abdomen, and raises the trunk off the ground by the arms, and gradually brings the weight over the thighs.

The child can crawl and move the legs fairly well, but the hind quarters swing about, and a light touch on the nates is sufficient to throw the child on to its buttocks. There is no lordosis. The spine is natural, and there is no deficiency of the laminæ. The knee-jerks are absent.

The electrical reaction.—The anterior tibial and peroneal muscles of both legs react readily to weak faradic currents. The gastrocnemii do not react so well, but the quadriceps extensor reacts fairly. The dorsal muscles of the back require a much stronger current. To galvanism—a fairly strong current is required to cause a contraction, and in the anterior tibial muscles the KCC = ACC.

Remarks.—These cases would seem to correspond with the conditions of muscular atrophy occurring in young children described by Werdnig and Hoffmann, and although in some points they differ from the description given, yet in the main they closely correspond. The chief differences would seem to be in the longer course run by the disease in the members of the family here recorded; in the fact that there was but little affection of the muscles of the neck; and in the absence of any true reaction of degeneration such as has been described in some of Werdnig's cases. It seems not improbable, however, that there are many cases which, clinically, do not conform to the description given by Werdnig and Hoffmann, and yet pathologically may be identical.

IV.—*Sequel of case of Leprosy shown in May*, 1900, *at the Clinical Society, showing the results of treatment by Ol. Gynocardii* (*Chaulmoogra oil*). By T. D. Savill, M.D. *Exhibited October* 24, 1902.

THE patient was a boy æt. 16, who was born in Demerara, and came to England when eleven years old. The disease had started at ten years of age with slightly raised erythematous and pigmented patches, most of which were anæsthetic. When last exhibited the patches were no longer raised, but over arms and legs and face were many pigmented, ovoid patches. Both ulnar nerves, and especially the left, were thickened. At the present time all anæsthesia and nerve-thickening has gone, and all pigmentation and erythema has disappeared. There only remain slight indications on the face, which are hardly visible to the naked eye.

The treatment consisted of Ol. Gynocardii, which was begun in August, 1899, ♏iv in capsule t. d. The dose was increased, and in May, 1900, he took fifty capsules *per diem*. At present he is still taking twenty capsules *per diem*.

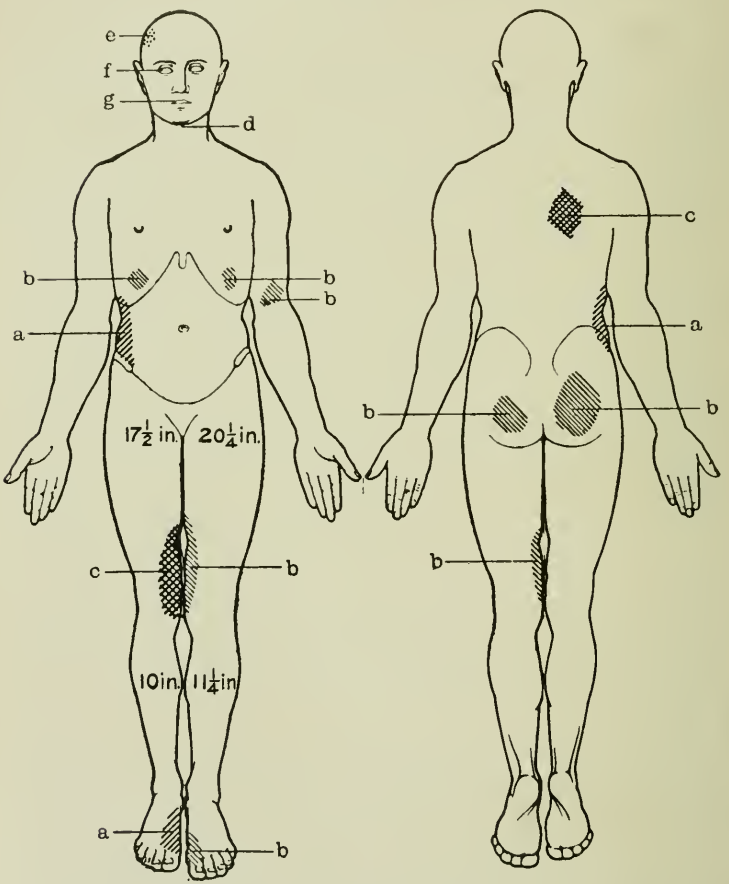
V.—*Case of Extensive Wasting of the type of Facial Hemiatrophy.* By Harry Campbell, M.D. *Exhibited October* 24, 1902.

WOMAN æt. 26. When aged twelve noticed right leg began to waste; the wasting has steadily progressed since then. At present it is smaller in all its dimensions than the left leg. On the inner part of the right thigh just above the knee, and on the dorsum of the right foot in the neighbourhood of the first interosseous space, there is discoloration with pronounced atrophy of skin and subjacent tissues. A similar discoloration, or mottling, is observed in the corresponding regions of the opposite limb. Similar mottled patches, sometimes with decided atrophy of the skin and underlying soft parts, are present in other parts of the body, *e. g.* on the right

upper lip, over the right scapula, and on the left side of the waist.

The right eyeball is retracted and the right pupil smaller than the left; it reacts to light and accommodation, and only slightly dilates under cocaine.

Fig. 11.



Over the right parieto-occipital region the hair comes out readily, and pain is sometimes felt here.

The tendon-jerks are active in the lower extremities; a doubtful Babinski's sign has been obtained in the right foot.

Sensation.—Besides the right-sided headache patient has

PLATE VI.

Illustrating Mr. Thomas H. Kellock's Case of Locomotor Ataxy with Atrophy of the Bones of the Feet.

Fig. 1.—Photograph of the feet showing the deformity and sinking backwards of the toes.

Fig. 2.—Skiagram of the right foot showing remarkable atrophy of the metatarsal and phalangeal bones.

Fig. 1.

Fig. 2.

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for the last few years had considerable pain in the right leg above the knee and over the anterior tibial region. There is no anæsthesia, but pronounced hyperæsthesia over atrophied patch on the right foot.

The electrical reactions are normal.

VI.—*A case of Locomotor Ataxy with Atrophy of the Bones of the Feet.* By Thos. H. Kellock. *Exhibited October* 24, 1902.

THE patient is æt. 40; he had rheumatic fever when aged eighteen. There is a history of gleet, but none of syphilis. His mother died of cancer. Three brothers and two sisters are alive and strong. He has been married seventeen years, but has no family. About three years ago he began to suffer pain in his feet, and had some ulcers on them, from one of which on the left foot a small piece of bone came away. Up to that time he had noticed nothing wrong with the shape of the feet, but from that time onwards this began to change, and they became shorter and deformed. In March, 1902, when much "run down," he had ulcers in the soles of the feet, and was taken seriously ill, with great pain in the abdomen and constant vomiting. This lasted ten days, and after a few weeks he went back to his work (manager of a restaurant), but has had to give this up again recently, as he feels so weak. He has suffered lately from hæmorrhage from the rectum, where he has hæmorrhoids. He also suffers from cough and "tightness" of the chest.

A truss which he has been wearing for inguinal hernia has caused an unhealthy-looking ulcer over the sacrum.

The patient has fairly well-marked symptoms of locomotor ataxy. The feet show old and recent trophic ulcerations, and most of the metatarsal and phalangeal bones appear atrophied and shortened.

VII.—*Displacement of the Heart due to long-standing Pleurisy.* By Raymond Crawfurd, M.D. *Exhibited October* 24, 1902.

B. M., æt. 15. Complains of cough and noisy breathing as long as she can remember. The cough has always been worse in the winter, and most troublesome when lying on her back. There has never been much expectoration, nor has it been fœtid. At eight months old she had a severe illness, said to be "dry pleurisy," which left her very thin and weak till three years old. At twelve years she had acute rheumatism.

Physical examination.—At times the face is distinctly cyanosed, and the finger-tips are a trifle blunt. The left side of the chest is hyper-resonant, and the area of cardiac dulness is absent. Stridor is felt and heard all over this side, gradually diminishing in intensity towards the base. The measurements of the right and left sides are almost identical. The movement of the whole right side is defective, and the intercostal spaces are narrower than on the left. Tactile vocal fremitus is much increased over the back of the right side, particularly below the sixth dorsal vertebra. A gurgling stridor is felt and heard over the larynx and right lung, most intensely at its apex. The percussion note over the summit of the right lung, front and back, seems normal. At the level of the fourth rib in front absolute dulness begins, and passes outwards to the level of the sixth dorsal vertebra behind; over this area, particularly behind, are heard cavernous breathing and loud gurgling *râles*.

The heart's impulse is best felt in the fifth right interspace in the mid-axillary line.

The larynx is normal, but the trachea appears to be slightly deflected to the right just above the level of the sternum, causing a prominence of the right side of the throat. The case is clearly one of collapse of the right lung, probably with extensive pleural adhesion. The lung parenchyma was irretrievably collapsed, and in its effort to expand to meet the chest wall it has tugged a bronchus widely open and drawn the heart right across the chest. These points are all confirmed by the appearances seen on transillumination of the chest. The case is remarkable for the degree of dis-

placement of the heart, the blunt end, presumably the right auricle, being seemingly in contact with the ribs in the right axilla.

VIII.—*A Child æt.* 11 *years* 10½ *months, from whom an Oval Tumour of the Right Kidney was removed ten years ago, on November* 15, 1892. *The growth with the kidney measured six inches in length, and was described as a Malignant Adenoma.* By J. D. Malcolm. *Exhibited October* 24, 1902.

THE history of this case is detailed in the twenty-seventh volume of the Society's *Transactions* (p. 94), and the patient was shown to this Society on April 22, 1895 (vol. xxviii). The kidney, with the tumour attached, is preserved in the museum of the Royal College of Surgeons (No. 3587 E). All the capsule of the kidney and some enlarged glands on the renal vessels were removed. Mr. Targett examined and described the tumour. It "was composed of tubes cut in various directions and closely packed together. They varied considerably in size, but there appeared to be no special arrangement of large and small tubes. The majority had a definite lumen, but to some (and these were chiefly the smaller collections of cells) the term tube was hardly applicable, as no lumen existed in them. The stroma of the growth was scanty and exceedingly delicate, save for a few strands of connective tissue which divided the field up into large loculi. The tubes which had a distinct lumen were lined with tall columnar epithelium, having one or two rows of oval or round nuclei at the base. The solid processes were made up of small spheroidal epithelium with darkly staining nuclei. One lymphatic gland, taken from the neighbourhood of the kidney, was examined, but the evidence of secondary deposit in it was inconclusive. The tumour may be classed with those known as malignant adenomata." No striped muscle nor any sarcomatous tissue was found.

The writer knows of only two other cases in which tumours of the kidney have been removed from children and the patients have lived two years after the operations. Both of these were operated on by Dr. Abbe, of New York. He

writes, under date October 13, that one of his cases was alive and well in the spring of this year, and there is no reason to doubt that she is alive and well now. In the other case the patient developed sarcoma of the opposite kidney three and a half years after the operation, and life was rapidly destroyed.

The patient shown has had no serious illness and has enjoyed good health, but has been rather paler than her brothers and sisters.

Three specimens of her urine were examined by the Clinical Research Association on October 25 and November 5, 1902, and March 6, 1903. The specific gravity was 1017, 1019, and 1026. The first specimen contained a little albumen. There was also a small amount of deposit in which there were "a slight excess of leucocytes and numerous clumps of tailed and polygonal cells with large nuclei. In addition there were a few red blood-discs and vesical squames and a very small number of calcium oxalate crystals." No casts were found, and there was no other abnormality. The other two specimens contained a trace of albumen, but were otherwise normal.

IX.—*Hypertrophic Cirrhosis of the Liver (probably of Syphilitic Origin) with Chronic Jaundice and Xanthoma.* By F. Parkes Weber, M.D. *Exhibited October* 24, 1902.

THE patient, Mrs. M. A. F., is a rather thin woman, æt. 54. There is very great uniform enlargement of the liver. This organ forms a rounded, tumour-like projection in the abdomen, is smooth and hard to the touch, and extends into the right iliac fossa. The spleen can be felt moderately enlarged. There is jaundice with a certain amount of dusky pigmentation of the skin, as in all cases of very chronic jaundice; also xanthoma, the latter tolerably symmetrical, but rather more on the right side of the body than on the left. Following is the distribution of the xanthoma:—A broad band along the margins of the upper eyelids; an area near the inner angle of each eye; some spots on each side of the face below the angles of the mouth; a large area on each side of the neck, and

scattered patches on other parts of the neck; a few spots over the upper front of the chest; the skin of the umbilicus; an area over the sacrum; some spots in each axilla; also *stripes* corresponding to the *folds of skin* below the buttocks, and to the folds on the flexor aspects of the elbows, hands, and great toes.

The patient was married at the age of twenty, and has had two miscarriages and eleven children, six of which are dead. Amongst the children still living is the last one, now ten years old and healthy. The enlargement of the patient's abdomen has probably existed for eight or nine years, but has become much more striking during the last two years. The jaundice has apparently only been noticed for about twenty months, and the xanthoma for four months. There is no evidence of alcoholism, but there are scars on the legs evidently due to syphilitic gummata. It is worth mentioning that several of the gummatous ulcers occurred twenty-five or twenty-eight years ago, apparently in connection with pregnancy, and are said to have healed under the use of a simple zinc ointment,—that is, without special antisyphilitic treatment. A dragging pain is sometimes felt in the right side, but there is nothing pointing to cholelithiasis. There is no family history of chronic jaundice. The degree of jaundice varies much from time to time. A great improvement has recently taken place in the patient's general condition, independently of any specific treatment, and the jaundice has become very slight. The causalgia of the hands and general pruritus have likewise much diminished. There is no evidence of any disease of the heart, lungs, or kidneys. The fæces are well coloured and the urine contains no bile, though on addition of nitric acid it becomes darker, and then spectroscopic examination shows two absorption bands, apparently corresponding to urobilin and uroroscin. Blood: hæmoglobin 90 per cent.; leucocytes, apparently not in excess; no abnormal kinds of corpuscles seen. Ophthalmoscopic examination shows no choroiditis or retinitis. The patient is now under antisyphilitic treatment. It is probable that the liver disease was started by syphilis. It is doubtful, however, whether the change in the liver should be regarded itself as chiefly syphilitic, or rather as a form of Hanot's disease started by syphilis, but perhaps in a person predisposed to biliary forms of cirrhosis (cholæmic diathesis of French authors).

X.—*Facial Palsy in association with Herpes Zoster.* By Stanley Barnes, M.D., M.R.C.P. *Exhibited October* 24, 1902.

Frederick G., a carpenter's labourer, was admitted to the National Hospital, Queen Square, on October 8, 1902 under the care of Sir William Gowers. He complained of a rash on the neck for seven days, and paralysis of the face for twenty-four hours. One of his brothers is insane, and his father is said to have been gouty and to have died of dropsy at the age of 57; the rest of the family history is unimportant. He had always been subject to colds, and was a moderate drinker. He was never of a "rheumatic" disposition. His occupation necessitated frequent exposure to cold, and on September 30 he was particularly exposed whilst working out of doors. The next day, whilst undressing, he found a few "pimples" on the right side of his neck. By the following morning the rash had spread, and on October 3 a profuse herpetic eruption was present over the right side of the neck and the right shoulder. The rash was quite painless, and did not inconvenience him, so he continued at his work. On the day before admission to hospital, six days after the onset of the rash, he found that the soap got in his right eye as he was washing his face, and on looking in the mirror found that his face was paralysed. No vomiting accompanied the disease, and a slight headache and some nausea were the only constitutional symptoms.

On admission to hospital, the day after the onset of the facial palsy, he was a well-built, healthy-looking man of 40 years of age. There was no sign of rheumatic affection. Smell and sight were good, and the optic discs were normal. There was no weakness of his jaw muscles, nor any herpes over the distribution of the fifth nerve. The left side of the face acted normally, but the right side was completely paralysed, the platysma also being involved. All the right facial muscles reacted well to the faradic current, quite as well as those of the left side; the galvanic reactions were also normal. His hearing was good on the left side, but he complained that everything he heard on the right side seemed muffled. He could hear conversation in a low tone as well with the right ear as with the left, but although he could hear a high-pitched tuning-fork well on the left side he could only hear

it slightly on the right. Bone conduction was equally good on both sides. The membrana tympani was healthy.

Taste was also somewhat impaired. The tongue was moist and clean, but he complained that since the face had been paralysed he could not taste properly, especially on the right side. Tested carefully, it was found that he could not distinguish quinine, salt, or sugar anywhere on the right side of the tongue, palate, or fauces, or on the right tonsil, whilst there seemed to be some impairment on the left side of the anterior two-thirds of the tongue, but none on the posterior third.

Over the right side of the neck was a rapidly healing herpetic rash, which had evidently been very profuse. Only a few vesicles remained, but the position of the former rash was easily defined. It had reached upwards as far as the ramus of the jaw, and behind as high as the mastoid process. From here the upper boundary curved inwards, reaching the middle line behind at the seventh cervical spine; the line continued thence to the second dorsal spine, from which it coursed almost horizontally outwards, crossing the spine of the scapula about its middle to reach the outer surface of the arm at about the level of the surgical neck of the humerus; thence the boundary line ran almost horizontally inwards to the middle line at the level of the second costal cartilage, and upwards from this point along the anterior border of the sterno-mastoid to reach the middle of the right ramus of the jaw. The skin over the area so enclosed was not anæsthetic, but a pin-prick was not felt quite so acutely here as elsewhere. No other abnormality could be discovered. Apart from the face the reflexes were normal; the urine had a specific gravity of 1022, and contained no albumen.

A blister was applied over the right ear and the face was faradised daily. He rapidly recovered, so that by November 3 hardly a trace of the rash remained, and all the movements of the right side of his face were again possible, though weak. By this date his taste and hearing had completely recovered. By November 28 hardly a trace of facial paralysis remained. Throughout, the electrical excitability of his facial muscles remained unaltered both to faradism and to galvanism.

This case is of considerable interest in that it was possible to carefully examine the patient at an early stage, and thus to discover certain details which materially assist in the localisation of the lesion. Several cases of the association of herpetic eruptions with local paralysis have been recorded,

and one of the most frequent of such palsies is that of the facial nerve. An excellent *résumé* of this subject is given by Eichorst (*Virchow's Archiv*, B. cxxxix, H. 3). He finds that from the cases he was able to collect the most frequent distribution of the herpes associated with facial palsy is on the same side of the neck and shoulder as in the case recorded here. Most of the patients are of the kind usually classed as "rheumatic," but nothing definite is known of the pathology of the condition. In most of the recorded cases no taste or hearing changes were found, but several of them were first carefully examined a considerable time after the onset, when perhaps some recovery had taken place. In the case recorded here, however, definite affection of taste on the corresponding side of the tongue, and of hearing on the same side, were observed, and the history suggests that they came on at the same time as the facial paralysis; they certainly improved very rapidly, and were normal again before the face had completely recovered. Assuming that they came on approximately at the same time, it is natural to suppose that they might be due to a single lesion; in other words, that the lesion which caused the facial paralysis had also involved in some way taste and hearing. The particular form of loss of hearing from which this patient suffered is that usually associated with paralysis of the stapedius muscle, whose nerve leaves the facial trunk near the geniculate ganglion; through this region are also coursing the chorda tympani fibres to supply the anterior two-thirds of the tongue. It is therefore possible that an acute lesion, such as an inflammation of the geniculate ganglion of the right facial nerve, had caused all of these conditions. Moreover it is now fairly certain that herpes zoster is the result as a rule of inflammation of the spinal posterior root ganglia through which sensory fibres pass from the skin involved; in this case the ganglia so represented are evidently the third and fourth cervical ganglia of the right side. Hence this association of symptoms strongly suggests that the patient was suffering from the effects of an inflammation of three ganglia, one motor (the geniculate of the facial nerve) and two sensory (the third and fourth right cervical root ganglia).

XI.—*A case of Congenital Syphilitic Ulceration of the Tongue.* By Lawrie McGavin. *Exhibited October 24, 1902.*

THE patient, a boy æt. 9, had developed a large pyriform ulcer on the dorsum of his tongue three days previously. His mother, who appeared to be a perfectly healthy woman, stated that a similar ulcer had appeared some months previously in the same situation, but had yielded to treatment in the course of a few weeks. There was an indefinite history of syphilis in the father. The boy himself had never suffered from any disease other than measles and chicken-pox.

On examination he presented a typically "adenoid" appearance, and his head was rather rachitic in shape. Beyond the presence of a slightly cracked condition of the left angle of the mouth, none of the characteristic lesions of syphilis were present, the teeth and eyes being normal.

The anterior third of the dorsum of the tongue was occupied by a large ulcer, pyriform in shape, measuring an inch by half an inch, the base towards the tip, and the stem towards the foramen cæcum. It was about half an inch in depth, the tongue being much thickened and increased in breadth. The edges were punched out, clean-cut, and raised, while the base was sloughy and of the colour of wash-leather. The surrounding tissue was intensely hyperæmic, but not markedly indurated, and the glands in the submaxillary region were moderately enlarged and of firm consistence. The ulcer had a tendency to run backwards along the fibrous septum of the tongue, and was not painful or tender to the touch. In the absence of any constitutional disturbance, or any symptom suggesting any of the usual causes of ulceration of the tongue, a diagnosis of congenital syphilis was made, and the patient was treated with potassium iodide to the extent of grs. xx *per diem*. At the end of a fortnight the ulcer had entirely lost its original characters, and measured one third by one quarter of an inch; the glands had almost disappeared.

XII.—*Case of Local Panatrophy.* By Sir W. R. Gowers.

THE remarkable case shown by Dr. Campbell has reminded me of a similar condition which came under my notice in private seventeen years ago.

Miss R., æt. 33, was seen in 1885. There was nothing relevant in her family history. Five years before, she had had a severe fall upon ice and was stunned for a few moments, but it seemed to leave no after effects. There was also a vague story of weakness of the spine some years before the fall, for which she had to lie on her back for a time. Three years before I saw her, weakness of the left arm was first noticed, and a year later some wasting of the shoulder. The right arm was found to be a little weak a year before she was seen, and the right leg six months later.

She was well nourished, but was said to have been stouter previously. There was no great loss of power; she could walk without a limp, and the grasp, though not strong, was proportionate in the two hands, R. 25, L. 19, K°. Her spine was straight, and nowhere tender. An apparent prominence of the last cervical spine at once attracted notice, but was found to be really due to the wasting of the soft structures below it, over the upper dorsal vertebræ, and especially to the left, between the spine and the scapula. Here was a hollow area, about the diameter of an orange, like a subcutaneous excavation, and over this the skin had a dusky aspect. It seemed due to a wasting of all the structures. To the right of the spine everything was normal. The muscles on the left of the spine below the affected area were a little smaller than those on the right side, but the difference was trifling. At first I thought that there was no electrical excitability in the affected area, but more careful observation showed that the difference was only in the strength of the contraction. The trapezius at the spot acted to the same strength of current as on the other side, but only very feebly.

A similar but slighter area of wasting existed over the posterior part of the left deltoid, where both the muscular and subcutaneous tissue had diminished so as to make the spine of the scapula, the head of the humerus, and the acromion stand out conspicuously. Over the middle of the deltoid the conditions seemed normal. Here also the elec-

trical reaction was very slight, and yet normal in degree, and the polar reaction to voltaism was that of health.

On the right forearm were two areas of similar change, although less in degree than on the back. Each was about the size of a walnut, and the skin over each had the same dusky aspect.

The only other part affected was the right thigh, where a similar small depression, of the same character and aspect, existed at the outer part, in the middle—at the junction of the vastus externus with the outer hamstring muscle; a small portion of both was apparently involved.

The only other symptom noted was that the right breast was smaller than the left, and that the skin over the region of the back adjacent to the affected shoulder had a curious mottled and uneven aspect, which I noted at the time to be as if the skin might previously have covered more adipose tissue. But the local position of the change makes this explanation improbable. I did not record any other observations on the altered colour of the skin, but I have little doubt that what I described as "duskiness" was really pigmentary.

I have heard no more of the case until to-day, when I telegraphed to the doctor who sent her to me, and received the reply, "Still alive and well. Has weakness of the right arm and complete right facial palsy." That shows that the malady has not been progressive. I will endeavour to obtain a fuller account of her present state.*

I headed my notes at the time, "Total local atrophy, analogous to facial hemiatrophy." From this, however, it differs in the random distribution of the affected islets. Yet the involvement of the foot in Dr. Campbell's case is perhaps a link between them.

Until more pathological facts are known it is hardly possible even to attempt an explanation of this remarkable affection.†

* This has been secured by the help of Dr. Stanley Barnes, whose account of the present state of the patient is on page 164.

† This case is referred to in the chapter on "Muscular Atrophy," in my 'Man. Dis. Nerv. Syst.,' vol. i.

XIII.—*A Case of Hemiatrophy Facialis (Morphœa or Localised Scleroderma of the Face).* By Dr. T. D. Savill. *Exhibited January* 23, 1903.

THE patient is a man, G. S., æt. 50. In 1880 he had a severe blow on the top of the head, and thereafter suffered from neuralgic pains in the vertex and face, sometimes of great severity, until 1895, when they subsided under treatment. In 1885 or 1886 he first noticed that the hair of the left side of the scalp and left eyebrow was coming off, and

Fig. 12.

that the face on the left side was becoming wasted. The wasting of the face at first gradually increased, but it has remained stationary for some years. There is no history of alcohol or syphilis. The patient has been a waiter or a "carver" all his life.

On the left side of the face the whole of the skin is shiny and smooth, and this, with the subcutaneous tissue, is considerably atrophied. The hair is almost absent from the left

anterior quarter of the scalp, and is also wanting in the inner half of the left eyebrow. The temporal and masseter muscles appear to be atrophied,* but the other facial muscles do not seem wasted. The electrical reactions, as far as can be ascertained, are not affected, and the masticatory powers are as good on the left side as on the right. The distribution of the facial atrophy corresponds roughly to the cutaneous distribution of the left fifth cranial nerve.

There are three other patches of atrophic skin upon the body, one upon the middle of the back about the size of the hand, which presents a few dilated venules characteristic of morphœa, and in this position there appears to be a slight tendency to excess of pigment. Another patch of atrophied skin may be seen behind the right ear, and a third over the outer side of the left knee. No history is obtainable concerning these, and, like the facial atrophy, they cause the patient no unpleasant sensations.

(Microscopic sections of the skin of the atrophic side of the face and of the patch on the back—showing atrophy of all the layers of the skin—are shown; also sections from another case of morphœa and of healthy skin for comparison. Photographs of the patient are also exhibited.)

XIV.—*Case of Ruptured Aortic Valve.* By Seymour Taylor, M.D. *Exhibited January* 23, 1902.

JAMES M., æt. 50, hammerman, was admitted to the West London Hospital on October 16, 1902, complaining of giddiness, precordial pain, and dyspnœa.

He had been in the army, and contracted syphilis seventeen years ago.

The patient was in usual good health until July, 1902, when, after a long day's heavy work, he complained of pain in the precordial area, and dyspnœa. The symptoms became gradually worse until his admission to hospital.

On admission he was found to have a loud aortic obstructive murmur and a louder diastolic murmur. The latter was musical,

* Some of those who saw the case, while admitting the notable atrophy of the skin and subcutaneous tissue, expressed doubt as to there being really any muscular atrophy.

of low pitch, about B below the staff, and was attended by a thrill. This murmur was so loud, and so wide in its distribution, that it could be distinctly heard at a distance of two inches from the chest, and with the aid of the stethoscope it could be heard all over the thorax, back and front, in the abdomen as low as the umbilicus, and as far down the upper limb as the middle of the humerus. It could also be heard by placing the stethoscope on the cranial vault.

There was no evidence of pulsatile tumour to suggest the existence of an aneurysm. He had no dysphagia and no cough. The vocal cords acted symmetrically, and his radial pulses were equal. The left ventricle was hypertrophied and probably dilated; the urine free from albumen and sugar.

The diagnosis of ruptured valve is made on the following grounds, viz.:

(1) A history of syphilis.
(2) The sudden oncoming of urgent symptoms.
(3) The patient's occupation.
(4) The musical diastolic murmur.
(5) The presence of marked diastolic thrill.
(6) The absence of any other definite signs of aneurysm.

The exact nature of the rupture can only be conjectural, but I am inclined to think that it is a perforation rather than a complete separation of a cusp from its attachments.

XV.—*Case of Removal of the Scapula of a boy æt. 9 years.* By Stephen Paget. *Exhibited January 23, 1903.*

SIDNEY C., æt. 9 years, thin and delicate-looking, but healthy. Swelling first noticed one month ago; no pain; no history of injury. There is a hard, nodular, rounded mass springing by a broad base from the right supra-spinous fossa, and passing forward beneath the clavicle. No signs of pressure on the vessels and nerves of the arm. No enlarged glands. Operation, April 30, 1902: excision of scapula; growth found to be cartilaginous. During operation transfusion of three and a half pints of saline fluid. Patient made a good recovery, and the movements of the arm are very good. Sutures removed May 12. Discharged from hospital May 21, wearing a support to prevent arm from dropping.

XVI.—*Charcot's Knee and Spontaneous Fracture of the Tibia and Fibula in the same limb.* By H. D. Rolleston, M.D. *Exhibited January* 23, 1903.

Widow, æt. 46 years, has had seven miscarriages, the last eight years ago. Lightning pains eight years ago. Visceral crisis six years ago. Five years ago sudden onset of

Fig. 13.—Charcot's knee and spontaneous fracture of tibia and fibula on the right side. (From a photograph by T. C. English, Esq.)

painless swelling of left knee after a long walk. Three years ago while going upstairs the left tibia fractured painlessly. Menopause came on a year ago, and was accompanied by

incontinence of urine. Altered character of voice noticed eleven months ago. On December 3, 1902, after diarrhœa lasting three days, lost power in right leg.

Present state.—Charcot's arthropathy of left knee, flail-like. Right knee unaffected. Early tabetic change in right foot, projection of the scaphoid bone from absorption of astragalus.

The left tibia and fibula about the middle show a projection the result of an old fracture; the union is very good. Paresis of the right leg. Absent knee-jerks on both sides. Flexor plantar reflexes present on both sides, more marked on right side. Complete paralysis of the left third nerve. Altered speech, occasional choking fits, is subject to attacks of faintness without any previous exertion; probably bulbar. Optic discs pale. Pupils contracted and do not react to light or to accommodation. The accompanying photograph was kindly taken by T. C. English, M.B., F.R.C.S., surgical registrar to St. George's Hospital.

Addendum.—This patient died on February 26, 1903, of wide-spread pneumonic consolidation, probably due to food passing into the lungs. The spinal cord showed well-marked tabetic degeneration. Dr. R. S. Trevor, who did the *post-mortem*, found on opening the left knee-joint much lipping of the bones with erosion of the cartilages; there was a quantity of very viscid, glue-like, synovial fluid in the joint. There were bony prominences from the scaphoid bones on the dorsal surfaces of both feet.

XVII.—*A Case of Traumatic Stricture of the Œsophagus; Gastrostomy two years afterwards (Albert's method), performed at the London Hospital.* By John R. Lunn. *Exhibited January* 23, 1903.

ROBERT C., æt. 63, a seaman, was admitted under my care on October 21, 1902, and gave the following history:— He had always enjoyed good health until 1898, when he met with an accident whilst at sea. He was removing the sheaves, weighing about 36 lbs., from the top-gallant yard of the ship, they being tied round his neck, and whilst coming down the rigging (top-gallant mast) a step of the ratlin broke, and whilst trying to save himself from falling the man gave his neck a violent jerk backwards, and the weight of the sheaves

seemed to press on the front of his neck. Two years afterwards (1902) he began to lose weight, and experienced some difficulty in swallowing, which enabled him to take liquid food only. He went to the London Hospital, where he was X-rayed several times, and a small œsophageal bougie was passed with some difficulty through an obstruction about $6\frac{1}{2}$ inches from his teeth. About two months after this Mr. Mansell Moullin performed gastrostomy, and he was discharged four weeks afterwards, when he returned to sea, feeding himself by the abdominal opening into the stomach. After the man's arrival in Boston, U.S. (which was about fifteen months after the operation), the wound healed, so he went into hospital there and had it reopened, and was then able to feed himself once more. In 1902, on his way to France, the wound began to heal again, and as he could not pass the tube he tried to dilate the orifice with a piece of wood, which caused pain and bleeding; this apparently caused some local peritonitis. The patient states that on his arrival at Havre in September, 1902, he went into hospital, when the surgeon trimmed the edges of the wound in the abdomen, which they stitched together and closed. When he arrived at the St. Marylebone Infirmary in October, 1902, the stitches were removed and the wound was healed. Since his admission he has gained in weight, and has been able to take solid food, though he gets some dysphagia now and then, and he has still some obstruction (spasmodic) of the œsophagus. About seven inches from the teeth he has some doubtful thickening round the larynx. The patient seems very well in health with the exception of the ventral hernia, which causes him some pain occasionally, for which he wears an abdominal support.

XVIII.—*Persistent Slight Jaundice of four years' duration in a Girl æt.* $18\frac{1}{2}$ *years, without obvious alteration in size of Liver or Spleen, and without marked Impairment of the General Health.* By F. Parkes Weber, M.D. *Exhibited January* 23, 1903.

THE patient, Kate C., born in London, the youngest child of Polish Jewish parents, is a fairly plump girl, with persistent slight jaundice, best marked in the sclerotics of the

eyes. The mother, who is living and healthy aged sixty-three years, had thirteen children altogether, of whom eight died early, and two miscarriages. Following is the sequence:—No. 1, still living; No. 2, dead; No. 3, dead; then the first miscarriage; No. 4, still living; No. 5, dead; then the second miscarriage; No. 6, still living; No. 7, dead; No. 8, dead; No. 9, still living; No. 10, dead; No. 11, dead; No. 12, dead; No. 13, still living, the present patient. The father is said to have died at the age of fifty-three years of "asthma." The patient's jaundice was first noticed when she was fourteen years old, on her return from a visit to Yarmouth, and since then she has had a tendency to slight bleeding from the nose and gums. Recently she has been subject to headaches. She has had no other definite illness that she knows of; never rheumatism, never scarlet fever. Menstruation commenced at about seventeen years, but has been irregular and often absent since then.

The patient has been under observation since the beginning of November, 1902. At about that time there was slight irregular fever, but with rest in bed, diet consisting chiefly of milk, and a little chlorate of potassium internally, this passed off in little more than a week. There is no obvious enlargement of liver or spleen, and, indeed, by physical signs nothing abnormal can be detected in any of the thoracic or abdominal organs. The gums have sometimes been slightly swollen, and have sometimes bled; and a few purpuric spots were on one or two occasions noticed on the limbs. The fæces have generally been well coloured, though sometimes pale. The urine (daily quantity about 1600 c.c.) is free from albumen and sugar. It has varied in colour between different tints of sherry, but is generally not very dark; it becomes darker on the addition of nitric acid,* and sometimes by the filter-paper method a Gmelin's reaction has been obtained. Blood (November, 1902): hæmoglobin 85 per cent.; under the microscope the red corpuscles looked normal, and the proportion of white to red cells was found little if at all increased; no abnormal forms of cells were seen. A second examination

* On December 15, when the urine was of specific gravity 1020, rather high coloured, and giving a Gmelin's reaction by the filter-paper method, Dr. Weber could detect no definite urobilin band by spectroscopic examination; but on January 26 this band was very distinctly present, even without the addition of nitric acid to the urine. On the latter occasion the urine was of specific gravity 1016, faintly acid in reaction, of a bright orange colour, and becoming only slightly darker on the addition of nitric acid; it gave a very distinct Gmelin's reaction by the filter-paper method. The specific gravity of the urine has been found to vary between 1010 and 1022.

of the blood (January 20, 1903) showed that the hæmoglobin reached 85—90 per cent. of the normal standard, and that the number of red cells in the cubic millimetre was 3,340,000, the white cells numbering 6720. The serum obtained from a blister gave a Gmelin's reaction. No evidence of any congenital deformity in the body can be discovered. There is no clubbing of the fingers. Since the patient has been under observation there has been very little change in the degree of jaundice, but the headaches have been less marked, and for a long time there has been no fever at all. She has gained weight in the hospital. The pulse-rate is generally about 80 in the minute. There has never been pruritus or xanthoma. Examination of the eyes by the ophthalmoscope shows nothing abnormal. Iodide of potassium in five-grain doses three times daily was given during nine days, but was discontinued owing to a pustular eruption; iodides disappeared from the urine two days after the drug was discontinued. On two occasions the patient was given a decigramme of methylene blue, and on both occasions the colour disappeared from the urine very quickly, once in about eighteen hours, once within two days. The ingestion of 100 grammes of glucose failed to produce any alimentary glycosuria.

The case seems to resemble those described by French authors under the heading "simple cholæmia," * but unlike some of this class, in the present case the jaundice is not congenital, and is not known to exist in other members of the patient's family. The pathogeny of such cases is not known, but the jaundice may apparently persist without leading to grave organic changes, as far as clinical examination goes, though the liver or spleen, or both these organs, may become temporarily or permanently enlarged. Occasionally there may be exacerbations of the jaundice or other symptoms, sometimes accompanied by pyrexia. Occlusion of one or more of the smaller hepatic ducts by a process of obliterative cholangitis is suggested by Dr. Weber as a possible explanation of the clinical features of these cases. The bile secreted from a small portion of the hepatic parenchyma would thus be poured into the lymphatics instead of into the bowel. In a case like the present one, however, it is almost impossible absolutely to exclude the presence of an atrophic form of cirrhosis. With reference to a suggestion

* The French term "Ictère acholurique" implies the absence of bile pigment from the urine, whereas bile pigment may sometimes be detected in the urine of these cases, and even when it cannot be detected its presence may possibly be masked by other pigments accompanying it.

kindly made by Sir Dyce Duckworth, Dr. Weber believes that an important subject for future observations is the part played by syphilis, congenital or acquired, in the production of obliterative cholangitis (present at birth or first appearing at any subsequent period of life).

XIX.—*A Case of Tabes Dorsalis in a Man æt.* 44. By Leonard S. Dudgeon. *Exhibited January* 23, 1903.

THE chief points of interest in the case, besides those which are common to the disease, are as follows:

(1) Tabetic disease affecting the astragalo-scaphoid joint of the right foot. The ankle-joint is free. (X ray.). Large

Fig. 14.

and hard bony prominence on the dorsum of the right foot. Duration five years. Onset sudden, with severe pain for twenty-four hours. No pain since.

(2) Atrophy of the small muscles of the right hand,

especially those of the hypothenar eminence and the interossei. Electrical reactions: Partial and complete R. D. in the atrophied muscles. Onset gradual. Duration about a year.

(3) Complete loss of the special sense of smell.

XX.—*Pneumothorax in a Child æt. 23 months; recovery.* By Percy Kidd, M.D. *Exhibited January* 23, 1903.

WILLIAM J., æt. 1 year 11 months, admitted to London Hospital on September 22, 1902.

History of cough, wasting, and refusing food for two weeks.

September 22.—*On admission* presented all the signs of right pneumothorax. No shifting dulness. Severe cough. Sputum offensive; no tubercle bacilli found. Irregular pyrexia.

October.—Signs of pneumothorax gradually disappeared. Breath-sounds returned; crepitation heard over right lung; slight dulness developed at right base.

November.—Localised empyema opened at the right base. Pus foul, cultures grew micrococci.

January.—Wound healed. Child steadily gaining weight.

XXI.—*Three Cases of Volkmann's Contracture.* By Leonard S. Dudgeon. *Exhibited February* 27, 1903.

CASE 1.—Child æt. 5. Fracture of right upper arm near the elbow-joint in December, 1900.

Treatment.—Plaster splints with the arm in flexed position. Within twenty-four hours fingers were swollen, cyanosed, and cold. Splints were not removed for four days, possibly owing to the absence of pain. Large pressure sore on flexor aspect of forearm. Hand and fingers in the typical position. Sensation normal. Electrical reactions practically normal.

January, 1903.—Shortened forearm, which is strongly pronated. The position of the hand and fingers is still quite typical. Movement has improved.

Treatment for two years.—Passive movements and massage daily. Faradism three times weekly.

Result.—Very slight improvement.

Case 2.—Boy æt. 9. Injury to the left forearm two and a half years ago. Anterior and posterior splints for three weeks. No pain. Arm and hand were cold and cyanosed. Fingers in typical position. Pressure sore. Several operations with no result.

March, 1901.—Patient was brought to St. Thomas's Hospital. Electrical reactions showed partial R. D. in the ulnar nerve and muscles, and also in the posterior interosseous group. Partial motor and sensory paralysis in these regions.

Treatment.—Massage daily.

February, 1902.—Electrical reactions normal. Fingers and hand as before, but no ulnar or musculo-spiral paralysis.

Operation by Mr. Clutton.—Resection of half to three quarters of an inch of both radius and ulnar from the posterior aspect of the middle of each shaft. Both wired.

March 25.—Bones united. Massage daily.

Result.—Great improvement.

Case 3.—Boy æt. 8. Injury to the left forearm in December, 1901. Anterior and posterior splints for five weeks. No pain when splints were removed. Arm was shrunken, fingers contracted, and sloughing sore on forearm.

September, 1902.—Child was brought to St. Thomas's Hospital. Hand and fingers in typical position. Electrical reactions normal. Scar on forearm. Skiagram showed no evidence of an old fracture.

October, 1902.—Mr. Clutton excised three quarters of an inch from radius and ulnar, wired ends.

November 1.—Good union. Massage daily.

Result.—Great improvement.

XXII.—*Dislocation of the Long Tendon of the Biceps Flexor Cubiti.* By H. Betham Robinson, M.S. *Exhibited February* 27, 1903.

PATIENT, æt. 27, had the head of the left humerus excised in St. Thomas's Hospital on September 19, 1900, by the late Mr. Anderson for recurrent dislocation. This had

occurred nine times in the preceding three years. The result has proved most satisfactory.

He came under my care at the same hospital at the beginning of this month, and gave the following account:— Six months ago he fractured the right clavicle, which united rapidly. On January 3 last he fell off his van, hitting the front of his shoulder against a sharp corner. He then noticed the arm could not be brought properly to the side, and was in the position it has assumed several times since. The condition has varied since the accident, being sometimes better and sometimes worse. The patient, however, himself speaks of it being dislocated like the other one was.

The position the arm takes is characteristic. The humerus is abducted and inwardly rotated to a slight extent; the shoulder-joint being then fixed, attempted movement is attended by rotation of the scapula. The head of the humerus can be distinctly felt in the axilla, and seemingly more prominent against the front of the capsule than normally. When it is "out" there is complaint of some tingling in the fingers generally.

The malposition can be easily rectified by, after fixing the scapula, bringing the elbow forward, thus relaxing the biceps, and then rotating outwards. The reverse can be demonstrated by placing the fingers in the axilla and then abducting, when something will be felt to slip, and then the joint is fixed.

The condition is apparently associated with an abnormal laxity of the joint capsule and an increased play of the tendon in its groove. In abduction with inward rotation the tendon slips over the great tuberosity, and at the same time the head of the bone is brought up against the front of the capsule at the anterior margin of the glenoid cavity.

Note.—The case, shown under the above title, did not prove so on operation. The biceps tendon seemed securely held in its groove, but there was a very thickened bursa over the great tuberosity in relation with the insertion of the supra-spinatus. The tuberosity itself was irregular and thickened, evidently due to osteo-arthritis.—H. B. R.

XXIII.—*Varicose Internal Saphena Vein in a Child.* By Thos. H. Kellock, B.C., F.R.C.S. *Exhibited February* 27, 1903.

THE patient is a healthy girl æt. 8 years; she has had measles, scarlet fever, whooping-cough, and chicken-pox, but no other serious illness.

A paternal aunt died of consumption, a maternal aunt of hip disease.

The enlargement of the vein was first noticed accidentally seven months ago, when it is said to have been of the same size as at present.

There is considerable enlargement of the right internal saphena vein at the upper part; its course seems to be slightly abnormal, passing rather far towards the inner side of the thigh. Varicosity is also noticeable at the inner side and front of the patella behind the internal malleolus and over the inner side of the foot.

Measured from the anterior superior spine of the ilium to the top of the internal malleolus, the right leg is half an inch longer than the left (R. = 23½; L. = 23). The circumference of the limbs is the same at all points.

Measured with calipers, the transverse diameters of the lower ends of the femora are the same ($2\frac{6}{16}$ inches.)

The veins have never caused pain or inconvenience. There is nothing abnormal detected in the abdomen or heart.

XXIV.—*Lymphatic Leukæmia with very acute onset of symptoms, or Acute Lymphatic Leukæmia.* By W. L. Ascherson, M.B., and H. D. Rolleston, M.D. *Exhibited February* 27, 1903.

LUCY O., æt. 7, admitted February 17th, 1903, suffering from debility, pallor, and shortness of breath.

Family.—One of five healthy children. Father reported healthy. Mother seemed healthy, has had several miscarriages. None of the children are prone to epistaxis. No malarial influence (none of the family have been abroad).

Personal.—Full-term child, breast-fed, always healthy until the present illness. Never known previously to bleed from the nose or to bruise readily. Never noticed to be pale.

Present attack.—On waking one morning (February 8, 903) mouth was seen to be drawn up to the left side; opening he mouth was difficult; fluid food introduced into the mouth dribbled away. Child seemed ill, dull, and apathetic. Next day, while playing, fell from a toy horse and hurt her shins. Mother was surprised at the *rapidity and extent of the bruising.* Child continued to be apathetic; grew increasingly pale and weak. Facial symptoms disappeared twenty-four hours after they were first noticed. The day before admission was seen in the out-patient department.

O.P. report.—Temp. 102·8°. Right side of neck painful. No lumps in the neck. Enlarged spleen.

Child returned home to come up for admission the following day. That evening she had violent epistaxis and bleeding (it was reported) from the throat, which continued all through the night.

State on admission.—Child fairly nourished, tired and apathetic-looking; distinctly anæmic. Some bruises on the shins, the result of trauma; one on the left arm unaccounted for. *No purpuric spots of any kind.* Slight bleeding (oozing) still from the gums. No stomatitis. No sign of epistaxis. A small conjunctival hæmorrhage was noticed in the right eye. Tongue was clean. Teeth defective, but not carious. The breath was foul, and had the characteristic smell of decomposing blood. The *tonsils* were much enlarged, and the right showed evidence of hæmorrhage. There were enlarged glands on both sides of the neck (anterior and posterior triangles) and in the left axilla. The enlargement was only moderate, and the glands were freely movable. The glands in the groins were abnormally palpable. There was no manubrial dulness or evidence of enlargement of the thymus.

Chest.—Lungs, nothing abnormal. *Heart,* showed evidence of moderate dilatation.

Abdomen.—Showed moderate enlargement of the spleen; the organ was felt about three inches below the costal margin on inspiration; the edge was soft and rounded; notch not felt; the organ seemed of fairly soft consistence. Otherwise abdomen seemed normal. No friction heard over spleen.

Osseous system.—No evidence of tenderness anywhere on pressure over the bones.

Special senses.—Hearing perfect; vision perfect.

Nervous system.—Nothing abnormal detected.

Urine contained a large deposit of unpigmented lithates, and was very pale in colour; otherwise normal.

Blood—red blood-corpuscles.—Per c.mm. 2,320,000; some poikilocytosis and irregularity in size of the individual corpuscles. One normoblast seen in a stained film.

Hæmoglobin.—30 per cent.

Leucocytes.—Total per c.mm. 60,000, of which—

Polymorphonuclears . . .	3·3 per cent.
Hyaline leucocytes . . .	1·8 ,,
Lymphocytes	95·0 ,,

No other forms were detected. The lymphocytes were for the most part large; showed characteristic fraying of the edges. In some cases budding and great irregularity in shape. Many particles (free in the blood) of lymphocytic protoplasm.

In the week preceding exhibition before the Society, the child improved under increasing doses of arsenic, the blood count showing 42,000 white, with 98 per cent. lymphocytes.

After doses of chloride of calcium, the hemorrhages from the gums and throat subsided.

March 13.—Since admission, February 17 (about four weeks), the spleen has about doubled in size. This raises the question whether, during the nine days (February 8—17) which elapsed between the onset of symptoms and the date of admission, the spleen may not have increased from the normal to the size found on admission; whether, in fact, the enlargement of the spleen found on admission in this case is any evidence that the case is not one of acute lymphatic leukæmia. The fact that the spleen was so considerably enlarged on admission raised the question whether the disease might have existed in a latent condition for some time before the acute onset of symptoms. On March 13, the child's condition was extremely grave, and the parents decided to remove her from the hospital. Death occurred within a few days, but autopsy was possible.

XXV.—*Syringomyelia with Dissociative Anæsthesia, Trophic Joints, Muscular Atrophy, and Exaggerated Knee-jerks.* By A. E. Russell, M.D. *Exhibited February* 27, 1903.

A MAN æt. 50, with well-marked trophic arthropathy of the left wrist and carpal joints. Both shoulders creak on movement, and the left is apt to swell on attempting to work. The dissociative anæsthesia is distributed over both hands and arms, neck, scalp, and trunk down to the level of the fifth thoracic segment. There is considerable muscular atrophy of some of the intrinsic muscles of the right hand, especially of the abductor indicis. The knee-jerks are greatly exaggerated, but his gait is not spastic.

XXVI.—*Cerebro-spinal Meningitis, Optic Neuritis; complete loss of sight followed by partial recovery of vision of unusual character.* By W. Pasteur, M.D. *Exhibited February* 27, 1903.

CHARLOTTE U., æt. 22 years. Family history good. Previous health good; no evidence of syphilis. Chlorosis lasting nine weeks early in 1902.

Present illness, September 1, 1902.—Began with severe occipital headache, radiating forward to temples and down the neck. On September 17 severe pain in small of back, unable to move in bed; vomited on 18th and 19th, and on two occasions after admission. Shooting pains of varying intensity in arms and legs since onset of illness. Squint in left eye and prominence of both eyes on September 23. Sleeplessness from the beginning, numbness and tingling in fingers since middle of September. No convulsions or twitching at any time.

On admission, October 1, 1902.—No anæmia, marked paresis of arms and legs (electrical reactions normal). Rigidity of neck muscles; no retraction. Gait very unsteady and feeble, but not staggering. Knee-jerk absent on right, just per-

ceptible on left side. Plantar reflex normal. Kernig's sign present (October 10). Babinski's sign: flexor response (?). Sphincters unaffected. Troublesome constipation throughout illness. Much tenderness and pain over cervical spine and upper dorsal vertebræ. Headache constant but slight, worse at night. Head always bent forwards on chest.

Eyes.—Slight proptosis. Complete paralysis of both external recti. Pupils equal, widely dilated, and insensitive. Some ptosis of right upper lid on October 10, and of left on October 11.

Right fundus.—"Choked disc" (swelling = 6 dioptres), margins hazy; small hæmorrhages round margin; veins swollen; arteries obscured by effusion in places.

Left fundus.—Similar condition in slightly less degree, 5 dioptres of swelling; field of vision about normal.

Tongue.—Slight but constant deviation to the right during first ten days. Speech clear. Intellect unaffected throughout. Sensation normal. Temperature normal from first to last. Treated with full doses of iodide of potassium.

Began to improve after October 22. Sight totally lost at this time. Optic neuritis beginning to subside on November 4 with slight recovery of vision.

December 1.—Pupil reacted slightly to bright light, but she cannot tell light from dark, and does not see an electric light when held up close to her face; is able, however, to distinguish a hand or book, especially if moved to and fro.

January 19.—Marked atrophy of both discs. Can find her way readily about the ward, and recognises the shape of objects without much difficulty—better against a dark background than against the light; is still unable to see the electric light held before her face, or to be sure of the position of the windows. Has almost completely recovered muscular power, and is practically restored to health.

February 22.—On her return from the convalescent home vision has improved slightly. Can locate the ward windows correctly, and sees the electric light about as readily as a more feebly illumined object, but the perception is only momentary, and immediately lost unless the light is moved, whereas her perception of the hand gains in distinctness as she continues to look at it. Both knee-jerks are still absent; otherwise she is in good health.

Eyes.—Pupils react to light. Discs pale, edges blurred, retinal vessels small. External ocular muscles normal. Fields for white very contracted.

XXVII.—*Webbed Arm and Fingers, with absence of Pectoralis Major.* By Raymond Johnson, B.S. *Exhibited February* 27, 1903.

J. S., a boy æt. 6 years. The anterior fold of the right axilla is prolonged downwards in the form of a web which passes upwards from the inner side of the elbow to the chest at the level of the third costal cartilage, 2 cm. above the nipple. The pectoralis major is either absent or represented merely by a few fasciculi of the clavicular portion. In the free border of the web can be felt a slender fibrous cord ending below at the internal condyle of the humerus, and at its other extremity terminating in a muscular fasciculus attached apparently to the second costal cartilage (chondro-epitrochlearis muscle). There is impaired development of the whole upper extremity, and the four inner digits are webbed. The right scapula is smaller, and lies at a higher level than the left. Numerous cases presenting a similar combination of malformations have been recorded, especially in German literature.

XXVIII.—*Graves' Disease in Father and Son; history of passage of milky urine in latter case antecedent to onset of present symptoms.* By H. Batty Shaw, M.D. *Exhibited February* 27, 1903.

W. B., a railway inspector, æt. 46 years, has been suffering from weakness for the last four years. Exophthalmos of slight degree was noticed a little over two years ago, and a month or two later the neck was found to be swollen. He is a very nervous subject, and is frequently troubled with palpitation. There is tachycardia (pulse-rate 100 to 112), tremor of the hands, retraction of the upper eyelids, and von Graefe's sign is well marked; flushing of the face is common, and the skin generally is moist. The enlargement of the thyroid gland is larger on the right side than on the left.

E. B., clerk, æt. 21 years, has always been rather nervous, and there appears to have been for several years some pro-

minence of the eyeballs. Nearly two years ago he met with a rather severe bicycle accident, and since then has noticed marked palpitation, and he says the eyes have been more prominent since the accident. The pulse-rate is about 120 to the minute; there is marked tremor of the hands, especially the right. The eyes are prominent and equally so. There is enlargement of the thyroid gland; von Graefe's sign is absent. The skin is moist, and the apex-beat of the heart is diffuse. The urine is clear as a rule, but occasionally urates have been found in abundance. Specific gravity 1017—1026; acid; no sugar. Albumen is present, sometimes as a mere trace; sometimes a well-marked cloud is present. No other proteid nor fat can be demonstrated. He passes water three or four times at night.

Extract from 'Clinical Society's Transactions,' 1899, vol. xxxii, p. 216.—Dr. Sidney Phillips reported this case to the Society on October 28, 1898. It had been noticed that the patient passed during the night or on rising in the morning urine resembling milk in appearance; occasionally the urine deposited large gelatinous clots. Occasionally a cream-like layer was formed on the surface; specific gravity 1025—1030. "Microscopically it was seen to contain large, round, highly refracting bodies, which were dissolved up by ether. When ether was added to the urine it became clear, but on evaporation, in most cases, no greasy stain remained on blotting-paper. The microscope also showed the minute granules described by Gulliver as the 'molecular basis of chyle.'"

This patient was under Dr. Sidney Phillips's care for three years, and during the whole of this period milky urine was passed. There has been no recurrence of the symptoms since January, 1901.

XXIX.—*Pneumothorax; rapid recovery. Case shown at the meetings of February* 13 *and* 27, *and skiagrams of the* 12*th and* 25*th.* By Sir Hugh R. Beevor, Bart., M.D. *Exhibited February* 27, 1903.

MR. A. A., æt. 25, clerk. Height, 6 ft. 1 in. Weight, 9 st. 7 lbs. Of healthy family. A similar attack to the present four years ago the only previous ailment. On February 8 sudden pain when stooping and lifting a shovel of

PLATE VII.

Illustrating Sir Hugh R. Beevor's Case of Pneumothorax.

Fig. 1.—Skiagram taken on February 12.

Fig. 2.—Skiagram taken on February 25.

Fig. 1.

Fig. 2.

coals. On the 12th, chest expansion: right side, 1¼ in.; left side, ¼ to ½ in. The skiagrams here depicted show much movement of a semi-distended lung from expiration to inspiration. At this date and at the Society the following evening there was no trace of any unusual resonance on combined coin percussion and auscultation except at a small area near the sternum over the second interspace. On the 25th, chest expansion: right side, 1½ in.; left side, 1¼ in. At this date skiagrams showed air had been absorbed from the pleural sac. No fever and no signs and symptoms other than those of pneumothorax were at any time observed in this case. Mr. A. Reid kindly took these skiagrams.

XXX.—*Transverse Fracture of the Patella in a Limb previously amputated below the Knee.* By Charters Symonds. *Exhibited April* 24, 1903.

THIS man's leg was removed in the lower third for a crushed foot. Walking on crutches two months after he fell, and was readmitted into Guy's Hospital with a simple transverse fracture of the patella. The laceration of the capsule was not extensive, there was no grazing or bruising of the skin, and it was difficult to be clear whether he had struck the patella or not. The patella was sutured by wire in the open method. The joint was massaged in a few days, and at the end of a fortnight he could flex the knee to a right angle. The range of movement is at present complete.

XXXI.—*A Case of Excision of the Spleen for Rupture.* By Mr. Beaumont (introduced by Charters Symonds). *Exhibited April* 24, 1903.

THE early particulars of this case were published in the *Lancet*. The patient is exhibited to-night to show his appearance eleven months after the operation.

A wagon passed over him on May 20, 1902. The abdomen was opened immediately, when much blood escaped. The spleen was found completely ruptured, and was removed.

The abdominal cavity was flushed with hot sterilised water and the abdomen closed. On the sixth day after the operation fluid was found at the base of the left lung, and six ounces of bloody serum removed. His temperature at this time was 105°, and subdiaphragmatic abscess was suspected. An incision was made in the loin over the angle of the eleventh rib, and this bone resected. The stump of the spleen was easily examined, but no pus found. The examination of the blood showed large numbers of streptococci. He next developed pleural effusion on the left side, from which two pints of blood-stained fluid were removed. Antistreptococcic serum was injected, and on the fifth day of the injections the temperature fell to normal (but rose again next day), and the patient was conscious for the first time for nine days. All the wounds about this time became septic, and were swabbed with pure carbolic acid. On June 5 pus was found in the left pleura. Portions of the seventh and eighth ribs were resected, and the cavity evacuated. The temperature continued to rise to 104° and 105° daily. The cavity was washed out with weak lysol, but this gave rise to acute dyspnœa, and had to be discontinued. The continuous injection of a small quantity of oxygen gas into the cavity by a soft rubber tube was next tried. The temperature quickly fell, the discharge ceased, and general repair set in in all the wounds, and from this time he made an uninterrupted recovery. For a month the lymphatic glands were notably enlarged, and the pulse was never below 112. At the present time, eleven months after the accident, the patient appears to be in normal health.

XXXII.—*A Case of Separation of the Upper Epiphysis of the Femur.* By Graham Simpson (introduced by Charters Symonds). *Exhibited April* 24, 1903.

YOUTH æt. 18. No history of trauma, but history of pain in hip ten months ago. Complains now of pain in hip on walking far, and limps. Trochanter prominent, slight shortening, loss of rotation, and abduction; wasting of thigh. Skiagraphy shows separation of head of femur. Diagnosis: ? traumatic; ? caries sicca.

XXXIII.—*A Case of Deformity of the Femoral Head and Neck which supervened without the patient's knowledge.* By CUTHBERT S. WALLACE. *Exhibited April* 24, 1903.

Z. R., male, æt. 13, presented himself for medical examination with a view of joining a benefit society.

It was then noticed that there was a lateral curve, and that the right leg was 1½ inch shorter than the left.

There is a little limitation of movement. The great trochanter is raised, and the neck of the femur is felt to be thickened when grasped in an antero-posterior direction.

A skiagram shows that the outline of the head, neck, and great trochanter, as well as that of the bone about the acetabulum, is fluffy and indistinct. The neck is thick and irregular, while its angle with the shaft is diminished. The articular surface is mushroomed, and recalls the deformity seen in osteo-arthritis of the old.

The case seems of interest in connection with the question of those deformities of the femoral neck that are described under the term coxa vara.

XXXIV.—*A Case of Fracture of the Acetabulum and Displacement inwards of the Head of the Femur.* By CUTHBERT S. WALLACE. *Exhibited April* 24, 1903.

J. G. A., male, æt. 43, eight months previously fell a distance of thirty feet and struck the ground with his great trochanter.

He was treated with rest in bed. On his attempting to walk it was found that movement was extremely limited, and that to bring the foot to the ground the back had to be greatly arched.

At the present time there is considerable muscular wasting of the left lower extremity. The prominence of the great trochanter is wanting. Flexion and extension is the only movement to be obtained, and that is limited to a range of

movement between 20 and 45 degrees of flexion. There is no shortening.

A skiagram shows that the femur is intact, and that the head and bone forming the acetabulum have been driven inwards.

XXXV.—*Case of Myositis Fibrosa.* By F. E. Batten, M.D. *Exhibited April* 24, 1903.

G. W., æt. 6, the second of five children, the others being healthy. Till nine months old the boy was a normal child; mother then noticed that the back was growing out and the legs were drawn up. He is a poorly nourished boy, of fair intelligence; he sits in bed with legs flexed and back curved, the head flexed on the chest, and the face drawn down on the right side by the contraction of the right sterno-mastoid. The spine cannot be straightened, and the boy moves as a whole. The arms are flexed and cannot be fully extended. The legs are flexed on the abdomen and at the knees, and cannot be extended. The muscles are small and firm, but no ossification of them is present. The knee-jerks can just be obtained. Sensation is natural. All the muscles react to weak faradic current. Under chloroform no relaxation of contracted muscles occurs.

XXXVI.—*Cretin with Marked Cyanosis of Extremities.* By F. E. Batten, M.D. *Exhibited April* 24, 1903.

V. S., æt. 3 years 9 months, is the ninth of nine children; the others are healthy.

The cyanosis of the extremities was first noticed when the child was one year nine months old.

The child has now learnt to walk, and can only say a few words.

Typical cretinoid appearance. There is marked cyanosis

of the extremities, which varies considerably from time to time.

The heart-sounds are feeble and distant, but no murmur can be detected.

XXXVII.—*Case of Chronic Bulbar Paralysis in a Girl æt.* 22. By R. Murray Leslie, M.D. *Exhibited April* 24, 1903.

M. Y., a corset maker, æt. 22, living in Northamptonshire, was recommended to come to the Royal Hospital for Diseases of the Chest on the supposition that she was suffering from consumption. There were some bronchitic signs at the bases of both lungs, and on March 9 she was admitted as an in-patient.

Family history.—Both parents, four brothers, and one sister are all alive and well; no history of nervous affection in the family.

Previous history.—No previous illness of any importance.

History of present illness.—There has been no influenza, diphtheria, or other acute affection to which present illness can be attributed.

In June, 1902 (ten months ago), patient noticed inability to pronounce certain words and letters distinctly. This condition gradually became more pronounced until she practically lost all power of articulation. A few months later she found she was unable to protrude her tongue properly. About the same time she had some difficulty in swallowing solid food, and last January began to have regurgitation of liquid food through the nose. For some months patient has had to support the back of the head in order to prevent it falling backwards.

Present condition—Speech.—Patient cannot articulate any words distinctly. Seems to talk with her throat and nose in a hoarse, grunting sort of way. It is impossible to understand what she says.

Tongue.—Extremely atrophied, soft and flabby, with numerous transverse and a few longitudinal furrows on the dorsum. The tongue lies almost motionless in the floor of the mouth, and cannot be protruded beyond the teeth. Fibrillary

tremors are distinctly visible on the surface. Taste is unaffected.

Palate.—There is almost complete paralysis of the soft palate. There is very slight elevation on attempting to phonate; liquid food tends to regurgitate through the nose; a snorting noise is emitted on blowing out the cheeks with the lips closed; while the inflated cheeks at once collapse on removing the observer's fingers from the closed anterior nares. Irritation of the soft palate produces no retching, coughing, or other reflex action.

Larynx.—There is complete paralysis of the left vocal cord, which lies in the cadaveric position. There is abductor paralysis of the right cord, which can, however, be adducted so as to cross the middle line and meet its fellow on the opposite side. There is thus only partial aphonia. A quantity of mucus constantly accumulates around the orifice of the larynx, and which the patient cannot expel either by coughing or by hawking, which are both imperfectly performed.

Pharynx.—There is considerable difficulty in swallowing, the patient having to throw forward her head in order to swallow even liquid food. The dysphagia seems partly the result of paralysis of the superior constrictors, and partly due to the inability of the tongue to move backwards. There is a constant accumulation of saliva behind the tongue, and which hangs in festoons between the pillars of the fauces.

Lips.—The saliva, which accumulates in the mouth, dribbles away at night and smears the pillow. During the daytime the patient is constantly wiping away the saliva with her handkerchief. There is inability to whistle, and the paresis is also shown by her efforts to purse up the lips and show the teeth.

Face.—There is some weakness and atrophy of most of the face muscles on both sides. The eyes cannot be closed completely, particularly on the right side. There is manifest weakness of the elevators of the upper lip on the right side. Fibrillary twitchings can be seen in several places, particularly over the chin and at the corners of the mouth. The facial muscles react readily to a fairly strong faradic current. There is wasting of the masseter muscles.

Neck.—Patient has to support the head to prevent it falling backwards. There seems to be distinct atrophy of the bellies of both sterno-mastoid muscles. The trapezii are also slightly affected. The sterno-mastoids react to a strong faradic current.

General condition.—There is general emaciation, patient only weighing five stones. She feels, however, perfectly well in her general health. She is somewhat emotional, and her memory is slightly defective. The thoracic and abdominal organs are healthy, and the bronchitic signs have disappeared. She has lost six pounds in weight since coming under observation. This may be attributed in part to her dysphagia, which has lately become more troublesome. There has been a slight trace of albumen in the urine. There is no wasting or paralysis of the muscles of the trunk or extremities. The ocular muscles are unaffected. There is no evidence of cerebral tumour, amyotrophic lateral sclerosis, or progressive muscular atrophy. There is no headache, sickness, or optic neuritis. The deep reflexes of the upper extremity cannot be elicited, and there is no jaw-jerk. The knee-jerks are brisk, but there is no ankle-clonus. There is no affection of sensation, and special senses are normal. Patient has been treated with Faradism and injections of strychnia without perceptible benefit. Her condition remains more or less *in statu quo*.*

XXXVIII.—*Two Cases of Congenital Hypertrophy of a Lower Limb.* By Clive Riviere, M.D. *Exhibited April* 24, 1903.

Case 1.—Frances C., æt. 3½ months. A difference in size of the legs was first noticed when the child was two weeks old, and the deformity has been increasing ever since.

A very well nourished, breast-fed infant.

Measurements (taken April 14) :

	Right.	Left.
Length from perineum to internal malleolus .	7½ in.	6½ in.
Circumference of thigh at centre. . .	11½ ,,	8⅝ ,,
Circumference of calf at centre . . .	7⅝ ,,	6⅛ ,,

The right foot is about a quarter of an inch longer than the left. The enlargement is very symmetrical, and greater at the proximal than at the distal extremity of the limb; it is formed mostly of the soft tissues, but an increase in size of the bones also is shown by a skiagram. The other limbs are healthy, and there are no congenital tumours or other abnormalities.

* The family doctor (Dr. Gibbons, of Desborough) has since written announcing patient's death six weeks later from cardiac failure. He stated that the early symptoms were strongly suggestive of post-diphtheritic paralysis.

Case 2.—Emily C., æt. $4\frac{1}{2}$ months. Enlargement of the lower part of the left leg was noticed at birth. Enlargement of the thigh was not noticed till the child was two months old.

A very well nourished, breast-fed baby.

The left leg appears to be about a quarter of an inch longer than the right; the enlargement is most marked in the thigh.

Measurements (taken April 16):

	Right.	Left.
Circumference of thigh at centre . . .	$8\frac{3}{4}$ in.	10 in.
Circumference of calf at centre . . .	7 „	$7\frac{1}{2}$ „

There is perhaps some slight increase in size of the right foot also. The other limbs are normal. There is asymmetry of the cranium, causing a projection of the left side; the left side of the face is fuller than that of the right. No other congenital abnormalities are present save a capillary nævus on the back.

XXXIX.—*A Case of Herpes Brachialis with Deltoid Paralysis.* By S. Vere Pearson, M.B. *Exhibited April* 24, 1903.

J. H., a painter, æt. 52, was taken ill on February 16, 1903. On getting up on the morning of that day he noticed a sharp burning pain about the left arm, and inability to move the upper part of the arm. The following day an eruption appeared covering the left arm, the patient describing it as "clusters of watery blisters."

On March 11, since the loss of power persisted, he came to St. George's Hospital, and was admitted into the hospital under the care of Dr. Rolleston. On admission the whole of the left arm was covered with an herpetic eruption already partly healed. This eruption extended from the shoulder to the wrist, and was present on both aspects of the limb, being especially plentiful over the skin area corresponding to the terminations of the nerves from the posterior root of the fifth cervical segment. There was complete paralysis of the left deltoid and infra-spinatus muscles, with some atrophy, and partial paralysis of the latissimus dorsi. The other muscles of the left extremity were flabby and rather wasted.

The herpes gradually healed. The paralysis remained stationary for about a fortnight, the other muscles of the left extremity becoming more wasted from disuse. Then the affected muscles began to regain their power, and they continue to do so. These muscles have been having galvanism; massage has also been employed. The other muscles of the left arm have become at the same time firmer and stronger. The electrical reactions are now normal; they were not tested when the patient was first seen. Considerable neuralgic pain persisted up to about three weeks ago, but has now gone. There is no loss of sensation.

There are no signs of caries, myelitis, nor of tabes. There are no signs of lead or arsenic poisoning, nor of heart disease. Some arterio-sclerosis is present. Cases in origin and nature similar to this case are related by Dr. Taylor in the *Guy's Hosp. Reports* (vol. lii, p. 37), and by Dr. Farquhar Buzzard in *Brain* (1902, p. 299).

The patient still shows considerable loss of power and wasting in the left deltoid and infra-spinatus, and slight erythema where the herpes was distributed.

XL.—*A Case of Bradycardia.* By S. Vere Pearson, M.B. *Exhibited April* 24, 1903.

J. P. is a farmer, æt. 46. In August, 1901, whilst at work in a western state of America, where he has lived for a great part of his life, he got a sunstroke. Since that time his pulse-rate has ranged between twenty and thirty-six beats a minute. He was never seriously ill before. There is no knowledge of what his pulse-rate was before the sunstroke. He is able to get about, but has been unable to do any work for the last eighteen months because he finds himself incapable of any exertion.

He is a man of nervous temperament, muscularly flabby, at present in St. George's Hospital under Dr. Cyril Ogle's care. The heart beats usually twenty-six times a minute; sometimes it is as frequent as thirty-two, and other times as slow as twenty-four. The pulse wave is good and the tension rather high; the artery is somewhat thickened. Examination of the heart shows that the left ventricle is large, the apex-beat being under the fifth rib, just outside the nipple line; no

other abnormalities are noticed about the heart. The urine is healthy, its average sp. gr. is 1024, and it contains a normal amount of urea. All the other organs appear to be healthy except the teeth; these are very deficient, for there are only two molars and some front teeth. The patient has no pains of dyspepsia, nor is there any apparent difficulty in gastric digestion.

Since the onset of the attack the patient has been subject to syncopal attacks, at first as many as four or five a day, lately very seldom—only about six in the last year. There is a momentary dizziness and then complete unconsciousness, with rigidity but no paralysis, lasting four to eight minutes. These attacks apparently resemble those of "Stokes-Adams' disease," described by Osler in *Angina Pectoris and Allied States* (p. 70.)

[*Additional note* (June 16, 1903.)—During the last few weeks small abortive systoles have become manifest. Two occur between each full beat of the heart: they give rise to visible pulsation of the jugular veins, and sometimes to a ventricular movement just perceptible on inspection of the apical impulse beat.]

XLI.—*A Case of a Large Spontaneous Ventral Hernia.* By T. Crisp English. *Exhibited April* 24, 1903.

THE patient is a plasterer, æt. 62, recently under the care of Sir William Bennett in St. George's Hospital. About twelve years ago a small swelling appeared in the right semilunar line rather below the umbilicus, and was attributed by the patient to heavy lifting. It steadily increased in size, but caused no inconvenience until a year ago, since when its weight has prevented his working, and has caused pain in the back with occasional vomiting. The patient states that he has always been stout, has not suffered from a cough, and takes only a moderate amount of alcohol. A belt has been worn for the hernia since its first appearance.

On examination there is a very large ventral hernia springing by a broad base from the right semilunar line, and measuring seventeen inches vertically and thirteen inches

transversely. It occupies the middle line and hangs down onto the groins; it is resonant all over, and cannot be reduced at all into the abdomen. The umbilicus is not on the skin covering the hernia, but on the abdominal wall in the deep fold between the hernia and the abdomen on the left side. The patient was admitted into hospital to be fitted with a fresh belt.

XLII.—*Case of Multiple Rodent Ulcers.* By W. Bruce Clarke. *Exhibited April* 24, 1903.

Daniel C., æt. 37.

History of condition.—Two and a half years ago first noticed small red spot near outer canthus left eye, which gradually grew in size. Seven months later second spot occurred near outer canthus right eye, and increased slowly in size. About the same time another ulcer appeared on the forehead above the right eyebrow. This one has healed of its own accord, but has left a scar exactly similar to the scarring which has occurred round the ulcers underneath the eye. There is an ulcer on the inner side of his nose, and one on the under lip, and another on the side of his neck; but he cannot give any accurate account of the dates at which these appeared.

Present condition.—In addition to these six patches which have been already described, there are numerous pimples on his face. They vary from the size of a millet-seed to that of a split pea. As many as twenty or thirty of these can be easily counted. Were it not for the fact that he states the ulcerated patches all began with similar pimples, they would perhaps barely deserve attention, as they have the appearance of small sebaceous glands in which the ducts are blocked.

Two of these pimples have been excised and examined microscopically, as well as pieces from the six ulcerated patches above alluded to. They one and all exhibit the microscopic appearance of typical rodent ulcers, and are displayed under the microscope.

The patient is now undergoing electrical treatment with X rays four times a week, and is deriving considerable benefit from it. The ulcers are healing rapidly.

N.B.—The patient remained in hospital about six weeks after he was shown, during which time the ulcers completely healed; but the pimples, though they improved and diminished in size, and in some instances entirely disappeared, had not altogether vanished. The patient declined to remain any longer.

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